NERVOUS DISEASES

JOS. T. O'CONNOR

NERVOUS DISEASES

WITH HOMŒOPATHIC TREATMENT.

BY

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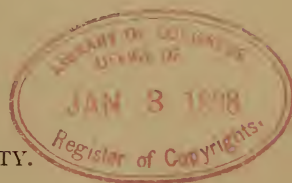MEMBER OF AMERICAN INSTITUTE OF HOMŒOPATHY,
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TO

THE MEMORY OF THE LATE

PROFESSOR A. R. THOMAS, M. D.

OF PHILADELPHIA,

AT WHOSE SKILLED HANDS I RECEIVED MY FIRST KNOWLEDGE OF THE
BENEFICENT POWER OF HOMŒOPATHY

THESE PAGES ARE AFFECTIONATELY INSCRIBED.

JOSEPH T. O'CONNOR.

PREFACE.

FOR a number of years past successive classes of students have urged the writer to issue in book form his own notes of lectures upon the diseases of the nervous system, given at the two homœopathic medical colleges in New York.

It is in response to these requests that the following pages have been put into book form. They are an amplification of the descriptive part of the lectures and a condensation of the anatomical details introductory to them.

The office of lecturer in a medical college is not only to give to his hearers a digest of the accepted text books on his subject, but also to note the advances made in his subject by investigators and clinicians the world over, and to select from these such results as seem worthy of acceptance for the advancement of the student.

Matters of purely academic or speculative interest have been omitted from the following pages except when their presentation would seem to conduce to a readier apprehension of related principles or to assist the better in memorizing important facts.

As the lectures are for students and practitioners of

homœopathy, other methods of treatment are not given except as allusions in cases not amenable to homœopathic medication as laid down in the Organon of Hahnemann. Hence the importance of diagnosis and a knowledge of the underlying pathological changes in any given case form a prerequisite to any attempt at cure. To apply homœopathic therapeutics from the symptoms of a disease admittedly incurable (except experimentally) is to do homœopathy a grievous harm.

Within its limitations Homœopathy is invincible; to enlarge its domain in even a slight degree is, it is hoped, not a visionary conceit, but in the writer's opinion is possible with accurate records of cases giving unassailable proofs of correct diagnosis and the use of corroborated clinical as well as pathogenetic indications for the use of our remedies.

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INTRODUCTORY.

TO medical students and indeed to many medical practitioners the term Nervous System carries the idea of infinite depth, of inextricable complexity and of insurmountable difficulty.

While these terms are perhaps truly applicable to certain parts and functions of the nervous system, yet so much is known of other parts and functions that the student of the nervous system is only hampered and discouraged in his work by having too prominently in mind the difficulties and complexities mentioned.

The nervous system is made up of parts morphologically alike in type and which perform functions of similar character, that is, to produce, to carry and to receive nervous impulses.

Such statement, however true literally, only continues the difficulty in studying the nervous system in health and disease. In illustration it may be said that all secretory organs have one function, that of secreting from the materials presented to them certain substances for certain definite purposes, yet no one would attempt the study of all the secretory organs as

members of one system merely because they all have the one common function of secretion.

Just as one secretion differs from another in composition and physiochemical activity, so nervous impulses differ among themselves, motor voluntary impulses being widely different from sensory involuntary ones and the latter differing again among themselves, so that an olfactory sense impulse may be utilized by the chemist as an aid in his analysis or an auditory sense impulse by the physicist in his investigations— yet all are manifestations of nerve-force.

Our knowledge of the specialized nervous functions of different nerve tracts and masses of nerve cells has been obtained largely by experimental methods on lower animals (atrophy method, method of development, method of secondary degeneration) but the results so obtained could not be applied to the human nervous system without the corroborative and confirming facts of pathological investigation in man, when accurately recorded symptoms and their progress during life were thus explained.

Notwithstanding such remarkable results, it may be said that the essential nature of disease is, except in cases of microbic or of toxic origin, still a mystery. Many diseases of the central nervous system are improperly so designated, as in invasion of the brain structure by a growth from without.

The neurone theory has given to the nerve cell and its neuraxone (axis cylinder process) and its dendrites (protoplasma processes) a *quasi* independence that has

long been foreshadowed. The mode of transmission of nerve impulses from a central neurone to a peripheral one is still a matter of discussion, yet the opinion most generally held is that there is actual contact of the end-branchings of the neuraxone of the first with the dendrites of the latter.

In actual practice the mattter may not be one of importance, yet any conception of the inter-relation of nerve units forming part of one system may offer at least a working hypothesis for the explanation of the ready action of correctly chosen highly attenuated remedies.

The independence of the nerve cell as an organ, while explaining many conditions observed in the course of certain nervous diseases, does not fully meet the question raised regarding the action of our attenuations, but the theory of the continuity of protoplasm throughout the whole organism, as elaborated by C. Heitzmann, and later by Bütschli, has been more recently advanced by a recent observer,* whose conclusions are summed up as follows: "The facts seem to warrant present belief that the living substance of all organisms is one physiologically continuous, living, plasma, homogeneous throughout in its intrinsic powers and properties, but having varied local and temporary habits of self-expression, which are largely and inextricably correlated with physical and chemical conditionings of its form and composition."

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* The Living Substance as such: and as Organism. By Gwendolen Foulke Andrews. Supplement to Journal of Morphology, Vol. XII, No. 2.

Through the agency of the continuous living substance we can at least picture to ourselves the undoubted action, often within a few moments, of highly attenuated drugs, but just as in the case of the higher vertebrates a limb or member when removed cannot be reproduced, so we must consider the nerve cell as an organ which, when destroyed, cannot be renewed.

Hence the necessity to the homœopath of an absolute diagnosis in order to determine just what part of the nervous system has undergone destructive change (as discovered by pathological investigation), and the limitations thereby set for restorative action through the influence of drugs.

The Hahnemannian view of the patient as a sick individual, and not merely as the bearer of some one ill-acting organ, is well set forth by the same investigator, who says that the "true organism is the invisible vesicular substance [continuous protoplasm], whose mass limits coincide of necessity with those of the living being; that all of the multiple parts and powers, functions and organs of living units are, and primarily were, of and for the substance as such, and only partially and incidentally for the animal or plant."

From the foregoing we can readily deduce the scientific reasonableness of Hahnemann's dictum to select the remedy according to the totality of the symptoms, and especially those of remarkable or peculiar qualities, the conditions of amelioration and aggravation of symptoms or other modalities, which

are the manifestations of the living, underlying continuous vesicular substance.

To select a remedy, especially in chronic disseases, the prescriber must have at his command something more than a Compendium or Condensation of our Materia Medica. The Encyclopedia and its Index or Symptom Register make the storehouse of our knowledge in this direction, with such additional provings as have since its publication appeared in our journals or in Transactions of the National and State Societies.

The homœopathic remedies recommended in the following pages (in italics) are in the potencies from 6th to 30th, except where otherwise specially designated.

To the writer the question of attenuation, as such, is of far less importance in curing disease than the Hahnemannian injunction to not repeat the dose as long as the previous dose is still acting. But in waiving discussion of the question of potency, the writer does not wish to be considered as justifying doses of tincture or of 1x trituration of powerful drugs, in amounts equalling the usual doses of the old school.

All adjuvants to medicinal treatment, included under the general term hygienic, should be employed. Their presentation and method of use do not fall within the scope of this book. But in this connection it may not be amiss to urge the physician to inquire concerning the use of tooth powders and

washes, toilet articles, mineral waters, etc., many of
which contain medicinal substances in amounts far
beyond those existing in our 3d attenuation, and
which cannot fail to introduce activities antidotal to
a remedy in use, or productive of new symptoms,
which, if not traced to their cause, must be mis-
leading to the strict prescriber.

The dosage and method of employing antipathic
or palliative remedies in cases confessedly incurable
are not given in the following pages; they will be
found in the allopathic works.

The systematic employment of electricity (in its
different modes), as subsidiary to homœopathic treat-
ment, must be studied in the text books of that
branch of medical science. Its use as a method of
diagnosis has, however, been given in condensed form
in the cases where such method is necessary.

For a more thorough knowledge of the anatomy of
the Nervous System than is given within the limits of
these pages, the student is urged to study Prof.
Edinger's Lectures on the Structure of the Nervous
Central Organs, or Prof. Obersteiner's Guide to the
Study of the Structure of the Nervous Central Or-
gans. Of both of these, American translations have
been published.

The Examination.

The functions of the nervous structures are so many and so various that a general outline of the methods to be followed in examining a case, to determine the presence or absence of a nervous affection, must be given in some detail.

Affections of the nervous system may be manifested by changes in sensation, whether of the peripheral sensibility or of the special senses; by changes in the motor functions of different parts of the body; by changes in the calibre of the blood vessels of a part, with consequent pallor in one case or redness in another; by alteration of glandular activity; by changes in the conditions of nutrition of the tissues and consequent atrophy or hypertrophy of a part; and in alterations in the *psyche*, that is to say, in the mental and emotional parts of our being.

Change in sensation may be a loss, complete or partial, or it may be an exaggeration of sensory impressions.

Loss of sensation is termed anæsthesia; when the strongest impressions are not perceived, the anæsthesia is complete, when they are but dimly perceived, so to say, it is incomplete; total and partial anæsthesia mean respectively that all, or a part only, of the body or of a limb is anæsthetic.

When used without qualification and in relation to

the periphery, the word anæsthesia refers to the ordinary sense of touch. But we have other peripheral sensations than those of touch. We can recognize cold and warmth, and we know what pain is. Whether a distinct set of nerve fibres and end organs exist in connection with the sense of pain or not, is a disputed point, yet it is known that pain sensations pass up the cord by a different pathway from that taken by the ordinary tactile sensation; in disease the former may be lost while the latter is retained.

There is no real hyperæsthesia as a real exaggeration of ordinary tactile sensibility. When we read of hyperæsthesia we know that algesia or pain sensation is meant—a touch is perceived as pain.

Analgesia means loss of the power of perceiving pain as such. In such cases the skin may be cut or pierced through without any painful sensation following, while the sense of touch is retained. When the power of perceiving painful stimuli is not lost, but is only lessened, we use the term hypalgesia.

Loss of the temperature sense, or thermanæsthesia, means loss of the power of recognizing differences of feeling produced by the application of a cold body and a hot one to the periphery. There is anæsthesia to cold alone or to heat alone in some cases, and there may be dysæsthesia of the temperature sense in which cold is felt as hot or *vice versa* or either may give rise to some different pain sensation.

The sensations of the parts beneath the skin have been greatly studied, yet with but little addition to

our knowledge. The peripheral mixed nerve fibres pass between muscles and through them and the cellular tissue, so that they must be affected in all tests such as that of estimating the amount of pressure made by a weight placed upon the hand lying on a table. Estimating the weight of an object held at arm's length may be by the effect on the nerves of the coverings of the joints, or by the amount of contraction of muscles needed to overcome the pull of gravity. So the sense of position of our limbs (without looking at them) tells us just where they are, whether straight or bent, etc. An experimenter can place one arm of his subject behind the back, fix the fingers and hand in some definite arrangement, and if the subject have the proper sensibility of the deep parts with no interruption of conduction to the brain, he can of his own volition move his other hand and fingers (still without the aid of the eyes) into the same relative position. This sense is called the muscular sense, and although the term has been attacked as being inappropriate and misleading, yet it gives a definite idea, and no better term has been proposed.

Loss of muscular sense is shown by ataxia, the latter word meaning absence of co-ordination. No voluntary action can be done by one muscle alone, several enter into producing a movement, and their shares in the extent of activity vary. To initiate a voluntary movement, or even such as has become automatic, the higher centres must know in just what amount of relaxation or contraction the required

muscles are, as well as the similar states in the oppo-
nents; then the proper impulse to contraction in the
one direction, and to relaxation in the other, being
made, the movement is accomplished not by stages
or by jerks of the separate muscles, but co-ordinately.
When, as in the ataxic, the information mentioned
above is not in possession of the higher centres, the
movement may be made, indeed, but it is done
awkwardly and with evident irregularity.

Ataxia may be of three kinds — spinal, cerebellar
and peripheral — the latter being as much due to
weakness of the muscles as to interference with sen-
sory nerve impulses. Spinal and cerebellar ataxia will
be considered in the sections on the spinal cord and
cerebellum respectively.

The prefixes hyper-, meaning excessive; hypo- or
hyp-, meaning diminution; ana-, meaning complete loss
and dys-, meaning perversion or falsity, may be used
with all the qualities of changed sensation.

At times in disease one touch may be felt as if
repeated—the prefix poly- is here used; at times a
touch made upon one side is felt as if made on the
other—this condition is termed allocheiria.

In testing sensation no special apparatus is needed.
A bit of absorbent cotton rolled into a loose wisp
may be lightly drawn upon the skin, the patient's
eyes being covered. He is then required to answer
if he has felt it and to say where.   If anæsthesia
appears to exist, the examiner's finger may be drawn
lightly over the spots or territory, and if still no sen-

sation is felt, then the touch may be made heavier. Anæsthesia to the cotton but not to the finger would be classed as hypæsthesia.

Next, the pain sensation is to be tested. A needle is used to lightly prick the skin; if no painful sensation is felt the needle may be passed deeper or finally through the skin. If sensation is felt at the first light pricking, of course no loss of the pain sense exists.

For testing the temperature sense two test tubes are used, one containing hot water, the other pieces of ice or, in the absence of ice, water as cold as can be obtained. Both test tubes should be dry on the outside. They are to be applied in irregular alternation and the subject is required to say in each case whether the hot or the cold tube has touched him. The hot water soon becomes cool in the test tube and requires renewal.

In all cases when testing sensation the eyes of the patient should be covered, so that he may not be misled into believing that he feels when he does not. When using the cotton or finger let him state first that he feels it, as the examiner should not ask "did you feel that?"

To poorly educated persons the word foot means often anywhere from the toes up to the knees; in such cases the subject may be required to point out the spot where the touch was made.

Areas of anæsthesia should be marked on the skin with ink or an aniline pencil and figured in the re-

cord.   All variations from normal sensation should be
recorded.

Paræsthesias are sensations that are entirely sub-
jective, that is, they are not the result of external
influences.   They are sensations of burning, prickling,
"pins and needles", freezing, etc.   They may be as-
sociated with other forms of abnormal sensation and
often precede the occurrence of anæsthesia.   The sen-
sation may be actual pain of the kind described.
Like everything wholly subjective there are no tests
for paræsthesia.

Alteration in motility of any part of the body does
not require an elaborate system of testing.   When
paralysis exists it is shown by inability of the patient
to move the paralyzed part.   The paralysis may be
complete or partial, the latter condition being indi-
cated by the word paresis.

Testing the strength of a limb or part is best
done by the examiner's own muscles.   For the upper
limb, the patient is told to grasp the examiner's first
three fingers and then to squeeze as hard as pos-
sible.   The same procedure is to be done with the
other hand.   Difference between the two will be
recognized by the examiner at once.   Even if both
sides are paretic the examiner can after a little
experience recognize the fact.

Next, the flexor power of the arm is to be tested.
If the patient flexes the arm as completely as he
can, the examiner putting his left hand on the shoul-
der in front, pulls down or extends the flexed arm

with his right. The amount of force requisite for this is inversely proportional to the amount of paralysis. Of course if there is total and complete paralysis the patient cannot flex the arm or can only make a feeble effort at doing so. Weakness of the flexors or extensors of the wrist is tested by overcoming the patient's effort to put the muscles in action.

The extension of the arm is tested by having the patient extend the arm fully, the examiner then trying to overcome it by doubling up the arm, if possible. The fingers may be tested in a similar manner, but more information will be obtained by requiring the patient to open the fingers, to separate them widely, to touch with the thumb successively the tips of the fingers of the same hand.

Testing the subdivisions of the lower limb may be carried out in a similar manner.

Testing the muscular strength is of greater value to the examiner in cases of paresis than in those of complete paralysis, the latter being self-evident on the patient's attempting a movement.

Hypertonicity of muscles is shown by rigidity of the limb, and the rigidity is difficult to overcome by the examiner at first; it does yield after some effort, and then the limb is easily bent. On straightening it again the weakness is evident until nearly in full extension, when extension is completed suddenly.

Tremor is a condition of alternate contraction and relaxation of opposing muscles of hand and arm, neck

and head, or leg and foot. The extent of motion is
small, and the recurrences from 4 or 5 to 10 or 12
in the second. If no tremor is evident, it may be
elicited by having the patient hold the hand out with
the fingers widely spread. The procedure will usually
bring out a tremor if it exist at all. Test the patient
further by telling him to hold the hand still; if the
tremor increases thereby, he should now be tested
by directing him to take from a table a glass of water
and taste it. If tremor begins with his taking up the
glass, and it increases in the extent of the oscillations
as it nears his mouth, he has what is called inten-
tional tremor.

The motions of chorea, athetosis and in general
convulsions need no test; in the latter, however, the
pupils should be examined, when possible, for the
reaction to light.

Contracture may be tested by endeavoring to over-
come it. In hysterical cases the examiner generally
finds a perceptible increase of the spasm acting
against his efforts. In organic contractures pain is
produced in the endeavor to overcome them. Con-
tractures due to anatomical shortening of unopposed
muscles cannot be overcome except by forcible rup-
ture of the muscle, or similar injury.

Paralysis, with atrophy, requires the employment
of electrical tests; these will be found described in
the section on electro-diagnosis. Atrophy is indicated
by evident loss of tissue, and alteration or sinking at
a part that normally is full. The relative part of

the other side may be used for comparison, unless it too has been similarly affected.

Loss of symmetry of the two sides of the face may be evident at first sight, or if slight, may require tests. Here, the patient is directed to separate the lips and show the teeth, to blow as if blowing out a candle, to close the eyes, to raise and then contract the eyebrows. Hemiplegia, peripheral paralysis of the facial nerve, hemiatrophy of the face, hysterical spasm of one side of the face, give different results with the above tests, which will be described under the proper headings.

Testing the special senses requires different tests. For taste, the four qualities, salt, sour, bitter and sweet, are employed, solutions being applied to the edge of the tongue on its anterior two-thirds. The substances used are simple syrup, a solution of common salt, extract of gentian, and very dilute hydrochloric acid. Application of each solution is to be made by a separate camel's hair pencil to the tongue, which is kept protruded until the taste sensation develops (or a reasonable time if it does not). The acid solution should be applied last, as it dulls the power of tasting. The patient is warned not to talk during the whole experiment, lest the solutions spread over the tongue, and when he notices a taste sensation, he selects from a card on which are written or printed the words sweet, bitter, salt, sour, the one that he has perceived. Each half of the tongue is to be tested by itself. (Goldscheider.)

Testing the olfactory nerve is done by holding a small vial containing some well-known odorous material, such as oil of peppermint or oil of lemon, before one nostril, the other being closed. On drawing in a whiff of it the odor will be perceived if the olfactory nerve is intact. Disease of the membrane may reduce or abolish the olfactory power. (Goldscheider.)

Tests in connection with eye and ear will be considered in treating of the respective nerves.

Electro-Diagnosis.

Although the subject of electricity in neurology in general is not within the plan of this book, yet the use of the electrical current in the diagnosis of nervous disease must be described with its methods.

When a current of electricity is passed through a motor nerve or muscle the related muscle contracts, as soon as the circuit is completed, if the current be of sufficient strength. A difference in results is noted when the current strength is increased and change of poles made. Normally, using galvanism, the negative pole or cathode being on the muscle, (the positive or anode on some indifferent part of the body) there will result contraction with less strength of current when the circuit is completed than when the poles are made to change places or when breaking the circuit. With increasing strength of current the following is the succession of contractions produced under the differences of arrangement just named. Cathodal closure contraction; anodal closure contraction; anodal opening contraction; cathodal closure tetanus. This series is expressed in tabular form and abbreviated as follows :

I. CaCC.

II. AnCC.

III. AnOC.

IV. CaCT.

The last term means cathodal tetanus contraction,
because with increasing current strength the contrac-
tions become more powerful and tend to prolong, until
finally they are here continuous or tetanic.

The strength of current causing a contraction
should be determined by a proper meter in the
circuit. As the unit of current strength is the am-
pere, and as only small fractions of an ampere are
used in medical electricity, the meter is arranged to
show thousandths of an ampere and hence is termed
milliamperemeter.

The electrode placed on some indifferent part of
the body when testing, should be of definite surface;
Erb's normal electrode has a surface of 10 square
centimeters. The differentiating electrode should be
small, Erb's being a series of three, spherical in shape
and graduated in size, the largest being about the
size of an average pea.

The electrodes, well wetted in warm water, being
placed in position and the negative on the nerve or
on the part of the muscle at which its nerve is nearest
the surface, (the handle of the differentiating elec-
trode should be provided with an interrupting arrange-
ment, so that the current can be made or broken at
the will of the examiner) a weak current is turned
on. As the current increases, contraction will be
noted in the muscles supplied by the nerve if the
differentiating electrode be over the nerve trunk; or
in a muscle if its motor point should have been selected.
Using the strength of current that brings the first

appearance of contraction, then by changing the pole by means of the pole changer the small electrode becomes positive and again the current is made and broken. If no contraction is observed it is clear that the negative closure contraction is greater than the positive, either closing or opening, which, as has been shown, is normal. If on the other hand, with the same current strength the positive give better contractions than did the negative closure, it is equally clear that the influence of the latter is less than that of the former—which is abnormal.

The faradic current, so-called, is in no wise different in nature from the galvanic. The secondary coil being generally used, the current is an induced one, and as it is only present when some change of current strength takes place in the original galvanic current derived from the cell which "runs" the faradic battery, a mechanism (the so-called vibrator or hammer) to produce very rapidly alternating makes and breaks is introduced. Contraction is caused by the secondary current just as in the case of the current from an ordinary galvanic battery, but before the muscle has time to relax a new flow of current comes and the contraction remains as long as the vibrator is acting.

Negative and positive, although existing in the faradic current, are not diffentiated in diagnostic use; but a better contraction is produced by the negative than by the positive.

When a motor nerve is severed the portion distal

from the point of section begins to degenerate, which process, according to Erb's investigations, is complete at the end of two weeks and in some cases earlier.

The electrical reactions have in that time altered in different ways. From the nerve, both faradic and galvanic contractility gradually lessen, until at the end of the second week they have disappeared. In the muscles supplied by the nerve, faradic contractility follows a parallel course, but often (not always) the galvanic contractility gradually lowers until near the end of the second week and then becomes increased and increasing reaches its apex at about the end of the third week; it then slowly falls, but still keeps above the normal, till about the end of the tenth week in cases that rapidly recover, to the end of the twentieth week in case of moderate severity, and in those that are not to recover it does not reach the normal level until the end of the fiftieth week. After these dates, respectively, the fall continues in the first until the end of the fifteenth week, when it begins to rise again; in the second it falls to the end of the fiftieth week and then slowly rises. While in the case not to recover it ascends to the end of the tenth week and then descends to finally disappear by the end of two years.

The accompanying figures from Erb show graphically the changes just described; in addition they show that recovery of a nerve after complete separation (or degeneration) takes at least six weeks from the beginning.

Fig. 1.

The first ordinate shows occurrence of the lesion and cessation of motility; the star indicates the return of the latter, the waved line indicates qualitative change.

Fig. 2.

Paralysis with late return of motility.

Fig. 3.

Incurable paralysis; no return of motility.

Fig. 4.

Partial RD; probably no degeneration of nerve.
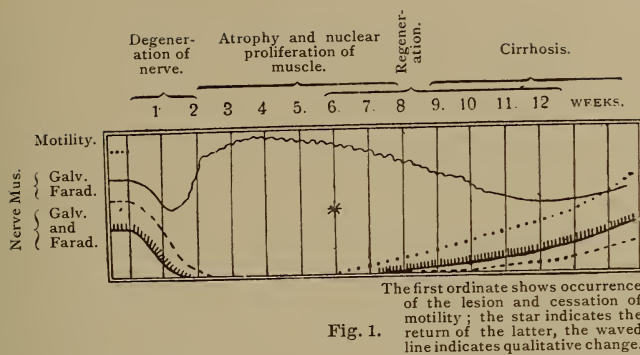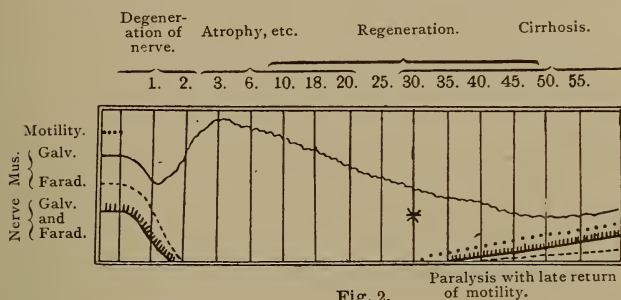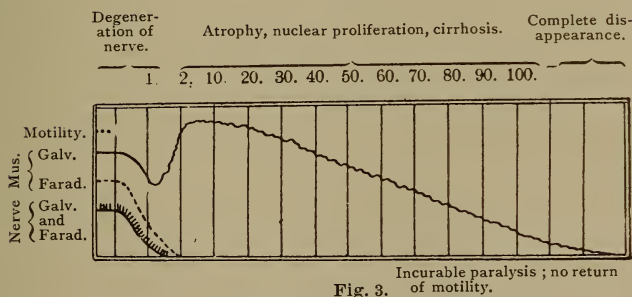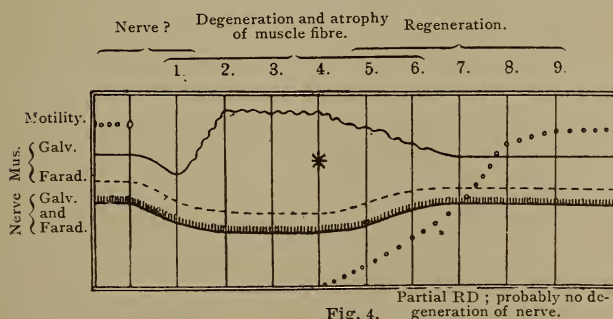
Degeneration of a nerve is shown by alterations in the contraction of its related muscle when the former is tested by either current; the contractions become weak and finally cease even with strong currents, reaction to the galvanic current continuing for a while after that to the faradic current has ceased. This loss of reaction through the nerve is termed an indirect quantitative change.

When the differentiating electrode is placed on a muscle supplied by a degenerated nerve it is found that instead of the negative pole giving the better response the positive often does so or at least may cause an equally good contraction. This is called serial or qualitative change.

Another typical change in the contraction of degenerated muscles is the absence of the quick, sharp, momentary character of the normal muscle's contraction, it being now slow and lagging. This is called a modal change and, if present, occurs with galvanic current only.

The reaction of degeneration (expressed by the letters RD) is therefore known by the existence of the changes just mentioned or, to recapitulate them: the muscle contracts slowly and sluggishly; it responds better to the positive than to the negative pole (or at least as well) and, indeed, at times in the earlier stages of the process, more readily to small amounts of current; and finally the faradic reaction of both nerve and muscle is lost.

A partial reaction of degeneration is said to be

present when, without any alteration of the normal polar succession, the contraction is weaker than normal and for both currents, and is slow and dragging. It is found in slight cases of peripheral motor nerve degeneration.

The motor points accessible on the surface have been marked on the body by Erb, Ziemssen and others. Because of their importance the figures and explanations are given on this and following pages.
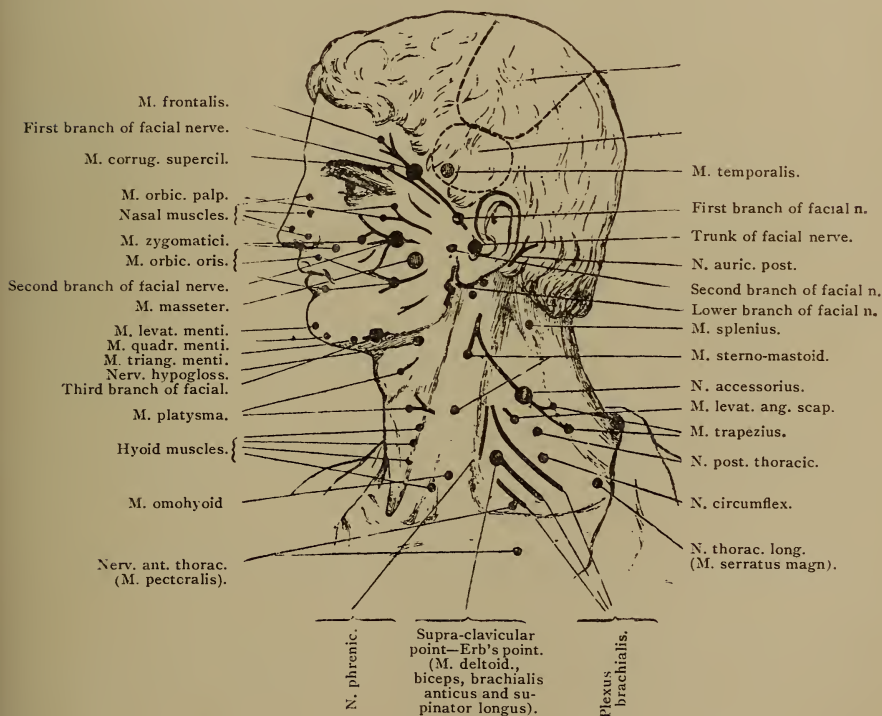
Fig. 5.

MOTOR POINTS OF FACE.

M. lumbricales.

M. lubricales.

M. adductor brev. poll.

M. flexor brev. poll.

M. opponens poll.

M. abductor brev. pollic.

M. opponens minimi digit.

M. flexor minimi digit.

M. abductor minimi digit.

M. palmaris brevis.

N. median.

M. flexor long. poll.

N. ulnaris.

M. flexor digit. sublimis.

M. flexor carpi radial.

M. flexor digit. profund.

M. pronator radii teres.

M. flexor carpi ulnar.

N. median.

M. brachialis ant.

M. biceps.

N. musc. cutan.

N. ulnar.

M. deltoideus ant.

Fig. 6.

UPPER EXTREMITY.—ANTERIOR ASPECT.

M. inteross. 1, 2, 3, 4.
(N. ulnaris.)

M. abduct. minimi
digiti.

M. extensor long. pollicis.

M. extensor brevis pollicis.

M. abductor long. pollicis.

M. extensor indicis.
M. extensor minimi digiti.

M. extensor communis digitor.
M. extensor radialis brevis.

M. supinator longus.
M. extensor carpi ulnaris.
M. extensor radial. longior.
M. supinator longus.

M. brachialis.

N. radialis (musculo-spiral).

M. tricps.

M. deltoid.
N. circumflex.
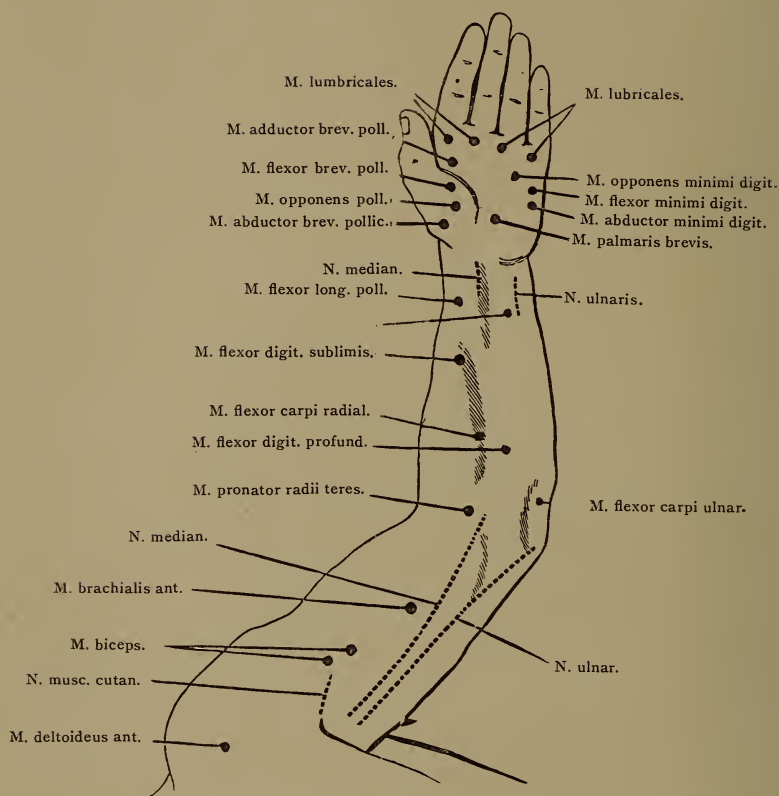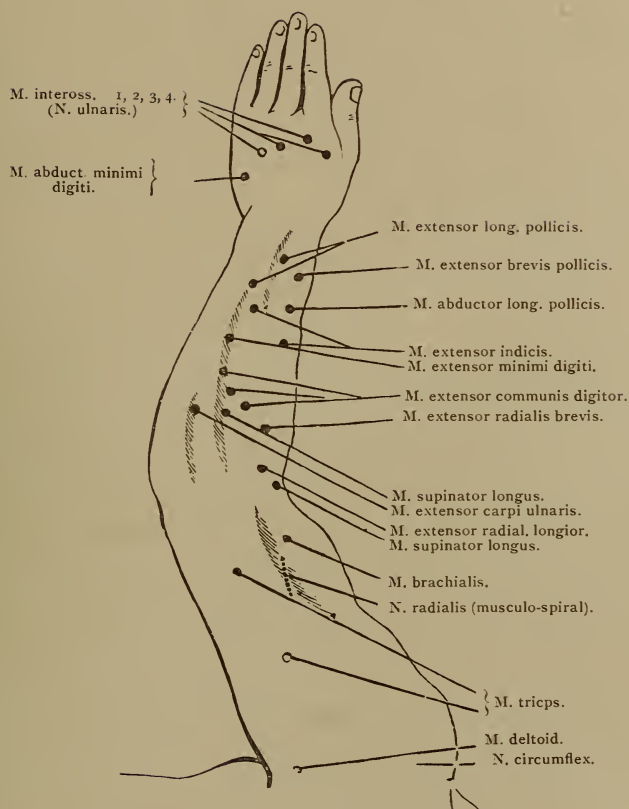
Fig. 7.

UPPER EXTREMITY.—POSTERIOR ASPECT.

Fig. 8.

LOWER EXTREMITY.—ANTERIOR ASPECT.
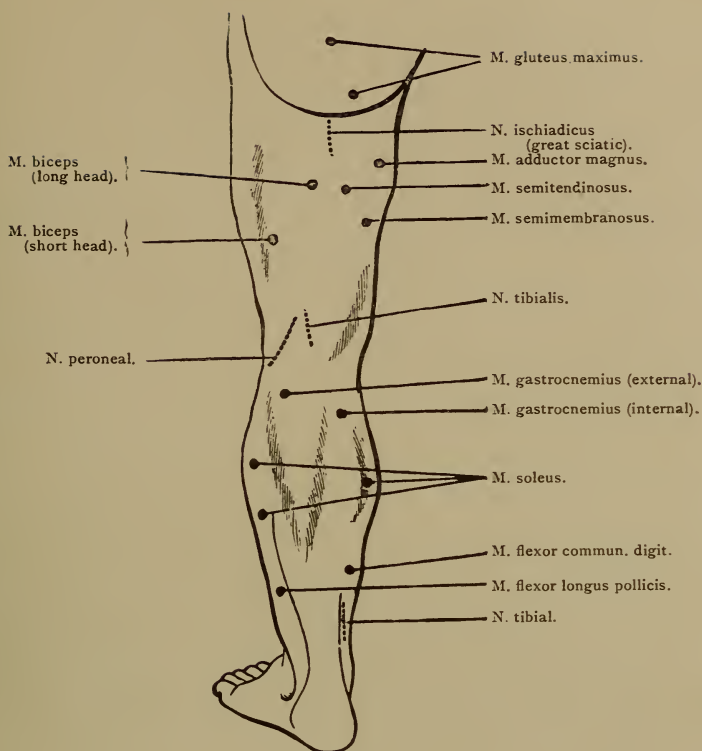
Fig. 9.

LOWER EXTREMITY.—POSTERIOR ASPECT.

Since degeneration of the central neurone does not pass on to the peripheral neurone, it follows that the reaction of degeneration excludes central disease, as it also does functional trouble. The site of the lesion may be the anterior gray horns of the cord (ganglion cells), the motor nerve roots or the nerve fibres. It is not present in primary disease of the muscles (muscular dystrophies) except late in the disorder. It is found in neuritis of all kinds, unless when very slight in degree ; in poliomyelitis anterior acuta, in

myelitis transversa if the anterior gray horns are
much affected; in bulbar paralysis, and amyotrophic
lateral sclerosis, in affections implicating, by pressure
or otherwise, the cells of the anterior gray horns or
those of the motor nerve nuclei in the medulla; in
syringomyelia, and after traumatic hæmatomyelia, and
in progressive muscular atrophy of spinal type. In
the progressive forms mentioned the reaction is usually
PRD rather than complete RD.

In prognosis the electro-responses of nerve and
muscle are of some importance. "Under like con-
ditions—i. e., in one and the same disease from the
same cause, the lesion is more severe, the duration
of the affection will be the longer, the outlook for
complete restoration will be the poorer the more com-
plete and typical the RD is and the more advanced
its stage. Thus, partial reaction of degeneration is
more favorable than the complete RD, and the later
stages of RD more unfavorable than the earlier ones."
—Erb.

Special reactions to electrical stimulation are found
in myotonia congenita and tetany. They will be de-
scribed in treating of those affections.

Peripheral Nerves.

Anatomy of Nerve Fibre.—Degeneration of Nerve.

Every nerve fibre originates in a nerve cell, and is, in fact, the extension of the axis cylinder process of the cell. Whether the prolonged axis cylinder ever divides in its course into two or more fibrils, although such view has been advanced, is not yet positively decided. In any event, the fibril soon after it leaves the cell receives a covering termed the myeline sheath, which covering is continued to the termination of the fibre.

Outside the myeline sheath there is on the peripheral nerve fibre another termed the connective tissue sheath, or sheath of Schwann. It is a delicate, yet dense, connective tissue envelope, which, at somewhat regular intervals in its length, undergoes constriction, thrusting away, as it were, in two directions the myeline, and pressing itself closely to the axis cylinder. At such points (constrictions or nodes of Ranvier) the axis cylinder can be seen, in a properly prepared specimen, uncovered by myeline. Between every two such constrictions is found a flattened, somewhat elongated, nucleus on the inner surface of the membrane. The sheath and its nuclei appear to be of the highest importance in determining the re-

generation of a peripheral nerve after injury or other lesion; the absence of this sheath and its nuclei in the nerve fibres of the central nervous system thus explains the irreparability of destructive changes in such fibres.
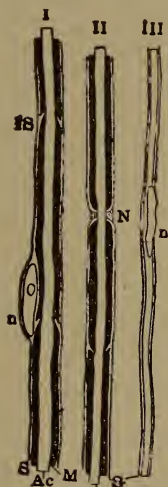

Fig. 10.

Fig. 10 (from Gowers) exhibits the structure of nerve fibre. I. and II. medullated, III. non-medullated fibres. Ac is the axis cylinder or neuraxon; M is the myeline sheath; S is connective tissue sheath; n is a nucleus of the latter; IS an incisure of Schmidt; N a node of Ranvier. The myeline sheath appears black, the effect of osmic acid stain.

The myeline sheath, after being prepared for microscopical examination, shows certain incisures, placed at an angle of about 45° to the long axis of the fibre.

These are termed incisures of Schmidt-Lantermann, and are believed to be non-existent in the living nerve, but their occurrence at somewhat regular intervals, and in definite direction, leads to the view that there is normally some structural differentiation at their sites.

The nutrition of a nerve fibre is kept up through trophic influence, or possibly by direct transfer of nutrient material from its originating cell. When a peripheral nerve fibre is cut across, the distal, or cut-off part, undergoes degeneration, most rapid in the interannular segment suffering the lesion, and next, in point of time, at the peripheral endings. The process of degeneration is a breaking down of the axis cylinder and the myeline sheath, the former disappearing, the latter being transformed into masses

or globules resembling oil, with granular debris, all finally disappearing. The nuclei of the connective tissue sheath increase in number, this increase, however, ceasing when the myeline within has been transformed into small droplets. There then remains only the collapsed connective tissue sheath.

Regeneration of the nerve occurs from the proximal cut end, the axis cylinder advancing along the course of the sheath of Schwann, the addition of the myeline envelope being slightly behind the progressing end of the axis cylinder. The process can be materially hastened in cases where a gap exists between the cut ends by suturing the ends together.

Consideration of the nerve cells and nerve endings is reserved for the section on diseases of the central nervous system.

Fig. 11.

Fig. 11 shows the degeneration of nerve fibre (from Gowers). At A the protoplasm and nuclei are increased, the myeline is breaking up, the process being more advanced near the nucleus n. In B the breaking up has gone on to the formation of globules. In C the latter are much smaller, and many have been removed, so that the fibre is narrow. In D all the products of degeneration have been removed from considerable tracts of the sheath.

Neuritis.

As the terminology of the word indicates, this means inflammation of a nerve. Theoretically, at least, any nerve may be the subject of the inflammatory process, but practically, certain peripheral nerves and plexuses are especially prone to inflammation. The causes of such inflammation may be arranged in three groups, *i. e.*, toxic, from without the body; auto-toxic, from within, and traumatic.

Traumatism, most commonly excessive or prolonged pressure, causes a localized neuritis, that is to say, in one or more nerves that have been thus subjected to the injury; the toxic, from without the body, and the auto-toxic, from within, may and usually do, involve in the neuritic process the nerves of more than one member; and when the toxic effect is not great or the resistance of the nerve to the morbid influence is great, a slight trauma may be sufficient to determine a neuritis limited to the part of the body so subjected to the latter influence.

The term peripheral nerve is often used indifferently for the nerve trunk and for the ultimate nerve fibres of which the latter is made up. And if the process of inflammation attack first the connective tissue about the nerve fibrils and the bundles of the same, the term interstitial neuritis is used; when the process is limited to the nerve fibrils themselves it is

said to be a parenchymatous neuritis. Clinically, no distinction between these two forms is practicable.

When a nerve becomes inflamed it begins to undergo a softening and alteration of structure both in its myeline sheath and in its axis cylinder; the inflamed spot thus undergoes degeneration so that here no nerve structure is discernible under the microscope. The portion of the nerve distal from this point undergoes degeneration even without extension of inflammation, according to the law of secondary degeneration, which tells us that any part of a nerve fibre if separated from its origin goes into degeneration. As the motor fibres of a peripheral spinal nerve take their origin in the large ganglion cells in the anterior gray horns of the cord, they degenerate outward from the point of the lesion. The sensory fibres of the same nerves arise from the posterior spinal ganglia and hence degenerate upward and downward. The older view that secondary degeneration goes in the direction in which the affected fibre carries its nervous impulses would thus seem to be not true of the peripheral sensory fibres.

As the cylinder axis of a nerve fibre is but the outgrowth and continuation of the cylinder axis process of the originating cell, the cell and its outrunning fibre being looked upon as a unit and termed neurone, it is evident that destruction of the cell is at once followed by destruction of the fibre. The effort has been recently made to prove that destruction of any part of a nerve unit or neurone is followed by de-

structive changes throughout the rest of the neurone and thus is explained an ascending neuritis even in motor fibres.

Not only is the nerve fibre proper dependent upon its originating cell, but also the muscle fibre to which the motor nerve fibre is sent and in which it ends, undergoes degeneration with the nerve.

There is therefore when complete degeneration of a peripheral spinal nerve trunk or a great portion of it has occurred, loss of function of all the affected fibres, that is to say, no impulses can be carried in either direction and paralysis of motion and of sensation results on the part of the affected nerve.

Since the nutrition of a muscle fibre is dependent upon the integrity of the nerve fibre ending in it, it follows that degeneration of the nerve fibre will be followed by a similar condition in the muscle cell. The latter becomes shorter and thinner, its striations more indistinct and closer together than normally; it takes on a peculiar glassy appearance (vitreous degeneration) and if regeneration of the nerve fibre does not occur the muscle fibre disappears. Meanwhile increase of the nuclei of the sarcolemma and connective tissue proliferation begin and in advanced cases the remains of a few muscle fibres in a mass of connective tissue are all that are left. The atrophy is usually evident to the eye in the case of superficial muscles.

In many cases of neuritis the lesion does not affect all the fibres or the process of degeneration is not

so rapid as that of regeneration; hence the paralysis may be slight in degree (paresis) for motion and sensation (hypæsthesia) or in the latter case there may be not destruction but irritation, so that paræsthesias occur in the distribution of the nerve fibres so affected.

From the foregoing considerations the diagnosis of a neuritis may be readily deduced. The paralysis or paresis, the anæsthesia, hypæsthesia or paræsthesia, or a combination of these, limited to the territory supplied by a mixed peripheral nerve, are sufficient to direct attention to that nerve as the organ whose functions have been interfered with or lost. Anæsthesia is, however, often surprisingly little in amount or limited in extent even after severe trauma, or lasts but a relatively short time. The explanation must be in the existence of other sensory nerve fibres for the same territory or a nerve anastomosis.

In an inflamed nerve whose continuity has not yet been destroyed pain and tenderness are common manifestations. The pain is constant, with variations in intensity, and in mild cases or those of moderate severity is described as a "dead aching", which by its persistence becomes torturing to the patient. It is increased by anything that causes pressure upon the affected nerve, such as muscular action in the part, passive motion, direct pressure, etc. In severe cases the quality of the subjective pain may be boring, tearing, burning, etc.

Tenderness to pressure along the nerve trunk is a

marked feature of neuritis, and even when in slight cases the pressure does not give rise to objective tenderness an increase of the subjective pain is often produced lasting for many minutes.

Paralysis is, as has been stated, one of the results of interruption in the continuity of a nerve, so that neuritis in all but the slightest forms gives rise to paralysis of the muscles supplied by the nerve affected. Thus neuritis of the musculo-spiral nerve is readily determined by the knowledge that the extensors of the forearm and the supinator longus are paralyzed. Examination of a patient should always include testing the different muscles as to their power of action under the direction of the patient's will.

A list of the muscles supplied by the peripheral (spinal) nerves, as well as statements of the action of those muscles, will spare the reader the trouble of studying out this information from his Anatomy should his memory of the facts be faulty.

Nerves of the Upper Extremity. Circumflex nerve. Supplies the deltoid, teres minor and the skin over the deltoid. In paralysis the upper arm cannot be raised, some slight amount of abduction is present from the action of the supra-spinatus. From the atrophy that occurs there is loss of the rounded contour of the shoulder by the prominence of the bony structures, and as the nerve supplies the structures of the joint the ligaments are relaxed. There is also to be seen and felt a gap between the head of the humerus and the acromion. Paralysis is to be differ-

entiated from anchylosis of the joint by the fact
that in the latter, if passive motion be made of the
upper arm the scapula moves also, not in paralysis.

Musculo-spiral nerve supplies the triceps, both sup-
inators, the muscles at the back of the forearm (ex-
tensors, abductors and adductors), extensors of wrist
and of first phalanges of the fingers, extensors and
abductors of the thumb.

In the majority of cases of paralysis of this nerve
the lesion is near the middle of the humerus, and
hence commonly the triceps is not affected. Because
of the extensor palsy there is inability to extend the
hand upon the forearm (the so-called wrist drop) and
also a projection at the back of the hand (synovial
sacs, bone) appears, and the first phalanges cannot be
extended. If the arm be straightened out the forearm
cannot be supinated. Paralysis of the supinator longus
is best shown by causing the patient to flex the arm
against an opposing force, when the normal bellying
of the supinator just below the bend of the elbow
will be found to be absent. Because of the loss of
the opposing action of the extensors the flexors can-
not act fully, and hence the grasp is weakened.

Median nerve supplies the pronator teres and pro-
nator quadratus, the radial flexor of the wrist, both
sets of flexors of the fingers, except the ulnar half
of the deep flexor, the long flexor of the thumb,
the muscles of the ball of the thumb except the ad-
ductor, the first two lumbricales and at times the
third. Flexion of the hand on the wrist is impeded,

and even only possible with a strong inclination to
the ulnar side; pronation of the forearm is impos-
sible beyond the mid-position to which it is brought
by the supinator longus. Flexion of second and third
phalanges of the fingers is lost but not completely in
the ring and little fingers. The thumb cannot be
flexed nor brought into apposition with the tips of
the fingers. The unopposed action of the interossei
on the last two phalanges causes hyperextension and
subluxation of them.

Ulnar nerve supplies the flexor carpi ulnaris, the
ulnar half of the deep flexor of the fingers, palmaris
brevis, muscles of the hypothenar eminence, the in-
terossei, the fourth lumbricalis and commonly the third,
and the adductor pollicis. In paralysis of this nerve
the power of flexion of the hand on the forearm is
greatly weakened, the little finger can scarcely be
moved, flexion of the first phalanges and extension
of the second and third are lost from paralysis of the
interossei, but not completely in the index and middle
fingers since these receive innervation from the median
nerve. Lateral movement of the fingers is impaired
and adduction of the thumb is lost.

The musculo-cutaneous nerve supplies the coraco-
brachialis, biceps and brachialis anticus muscles, the
chief flexors of the elbow. Paralysis of the nerve
prevents flexion of the forearm when in position of
supination, but flexion can be done when the arm is
pronated, by flexing the supinator longus.

Combined palsies of the nerves of the arm are

frequently seen and arise from various causes. Disease or injury above the brachial plexus, or affecting the plexus in part or entirely, or affecting more than one nerve trunk below the plexus, can readily produce varied combinations of paralysis. Severe pressure above the clavicle readily affects the fifth and sixth cervical nerves, as they begin to enter the formation of the brachial plexus. Electrical stimulation at a point two fingers' breadth above the clavicle and one finger's breadth behind the sterno-cleido-mastoid muscle causes contraction together of the deltoid, brachialis anticus, biceps and supinator longus (Erb's point: supraclavicular point). A not uncommon form of paralysis of the upper part of the brachial plexus occurs from pressure made above the clavicle by the accoucheur in extracting the delayed head or by prolonged pressure during parturition on the shoulder girdle, thus approximating the clavicle and spinal column. This is known as Erb's paralysis. At birth the peripheral nerve fibres are poor in resisting power and the effect of a severe lesion then is long lasting and may be irremediable.* The power of the

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* In the new born child and for several weeks after birth the resistance to both faradism and galvanism is very high as compared to that of the adult, varying from 7,000 to 10,000 ohms during the first week of extra-uterine life. In addition to this, the very young individual shows but little sensitiveness to currents, both faradic and galvanic, that cannot be borne by the adult. The contractions caused in the muscles by either current, whether direct or indirect, differ from those in the adult in being slow and dragging instead of being quick and sharp. The anatomical explanation of these long known facts is found by A. Westphal to be due to the imperfect development of the nerve fibre at birth and during the first few weeks, the myeline sheath being absent in spots and sometimes over long stretches of nerve axis, and when present being thin and evidently but poorly elaborated. The inutility of employing electricity as an aid to diagnosis in cases of Erb's paralysis during the first few weeks of life is thus apparent

Microscopical examination shows in the peripheral nerve in the very young, appearances very similar to those seen in the adult nerve degenerated after separation from its cell. The subject matter of this note is treated very thoroughly by A. Westphal, in *Archiv f. Psychiatrie u. Nervenkrankheiten*, Bd. XXVI., Hft. I.

muscles of the hand and lower arm is not lost, but
the upper arm is motionless and the humerus is ro-
tated inward, so that the forearm hangs with its ex-
tensor side facing forwards. This inward rotation is
stated by Goldscheider to be in the majority of
cases not due to paralysis of the external rotator, but
to separation of the epiphysis since the inward rotator
is inserted lower down than the outward rotator.

Another form of lesion of the brachial plexus is
seen when the injury occurs from below, in the axilla,
involving the eighth cervical and the first dorsal nerve
as they enter the plexus. The muscles of the hand
are paralyzed, the flexors in the forearm, and when
the seventh cervical nerve is affected, the extensors
of the hand are also involved.

Intermediate forms may occur or the plexus as a
whole be affected.

Besides the motor paralysis, oculo-pupillary symp-
toms may be observed when the roots are affected
before the giving off of the rami communicantes.

Dislocations, fractures, in the region of the shoulder
joint, forced retraction of the shoulders, the arms
being raised, during operations, direct pressure from
a badly fitting crutch in the axilla, violent muscular
activity as in throwing, etc., can produce injury to
the plexus, and a primary neuritis of the plexus has
been observed.

Sensory disturbances may be slight in degree, or on
the other hand quite marked after traumatic neuritis.

Nerves of the lower limb. Affections of the lum-

bar and sacral plexuses are far less frequent than those of the brachial, chiefly because of their protected situation, yet occasionally they, or some of the nerves from them, are the subject of special injury during prolonged parturition, by pressure of the forceps, or by tumors, etc.

Anterior crural nerve paralysis renders the patient unable to flex the leg at the hip joint except partly by the action of the psoas muscle, which is not supplied by this nerve; the extensors have no power, so that the leg cannot be extended upon the thigh. Hence standing and running are greatly impeded, if not prevented.

Paralysis of the obturator nerve causes inability to adduct the thighs; hence the legs cannot be crossed, nor the knees pressed together.

Paralysis of the superior gluteal nerve, a rare occurrence by itself, causes loss of power of internal rotation, abduction and extension of the thigh.

Paralysis of the sciatic nerve causes loss of power in all the muscles of the leg and foot, and the power of flexion of the leg upon the thigh is gone.

The peroneal or external popliteal nerve supplies the peroneus longus and brevis, tibialis anticus, extensor communis digitorum, longus and brevis, extensor hallucis longus. Its paralysis results in what is known as "drop foot", the foot hanging lax when the leg is lifted; the toes catch on the ground when the patient attempts to walk and the outer toes cannot be extended, the foot cannot be abducted nor dorsally

flexed and the outer edge of the foot cannot be raised. When the paralysis is of long standing the unopposed action of the calf muscles produces shortening and consequently pes equinus.

The internal popliteal nerve innervates the flexors, both long and short, of the toes, the calf muscles, tibialis posticus, adductor and abductor of the great toe, the abductor of the little toe, the interossei and lumbricales.

In paralysis of the plantar nerve flexion of foot and toes is lost; adduction is lessened; the patient cannot extend the foot nor raise himself upon his toes. If the paralysis be of long standing the unopposed action of mucles innervated by the peroneal nerve causes talipes calcaneus with hyperextension of first phalanges of the toes and flexion of second and third.

The areas of sensory distribution of the peripheral nerves will be more readily understood by reference to the accompanying illustrations than by any description.
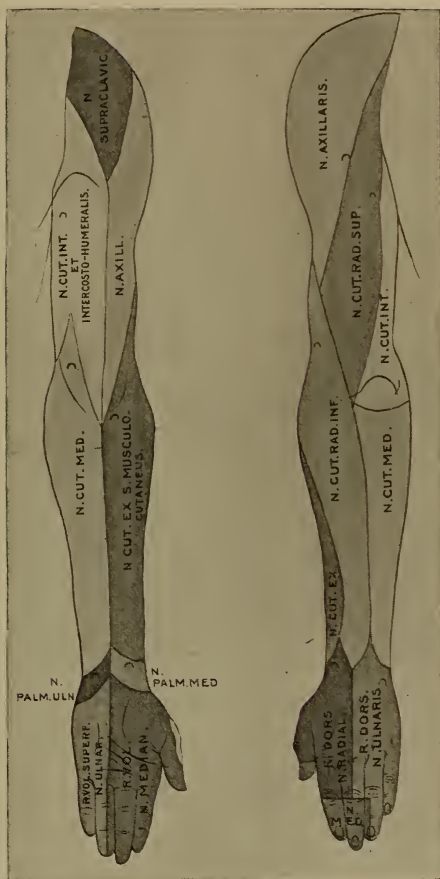
Fig. 12.

From Hesse. The small curves mark the places of exit of
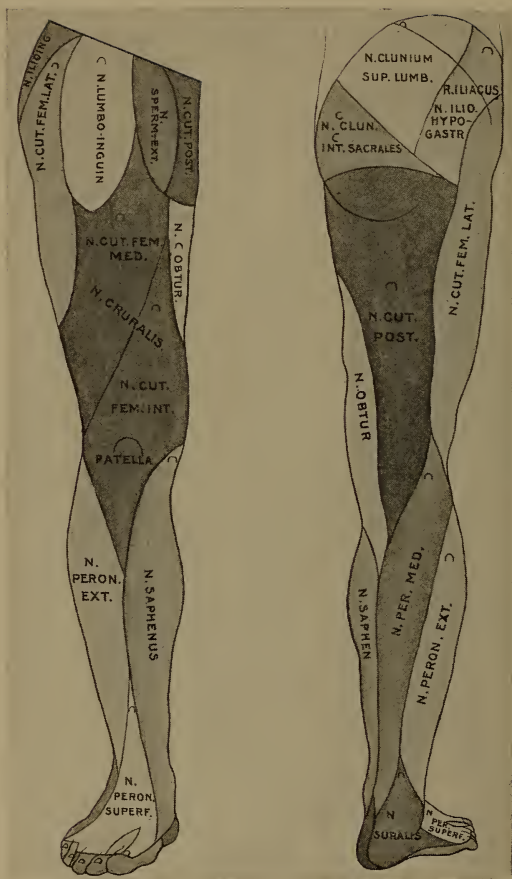nerve branches through the fascia.
N. axillaris =circumflex.

Fig. 13.

From Hesse. The small curves mark the places of exit of nerve branches through the fascia.

N. clunium sup. lumb.=gluteal distribution from lumbar nerves ;
N. clun. int. sacrales=gluteal distribution from sacral nerves.

Multiple Neuritis.

The distinction between neuritis arising from injury and a neuritis of toxic origin can usually be made out by eliciting the history of the case. Yet the writer has seen cases in which the history of traumatism was obtained only after repeated questioning. All the non-traumatic forms may be safely called toxic, either endogenous or exogenous, the exception being possibly where the localized influence of cold is the only assignable cause. In one instance a ride on a hot day in the open air produced a neuritis of the brachial plexus, and also of the dorsal nerves supplying the trapezius of the same side. General exposure to cold may cause suppression of cutaneous activity and consequent accumulation of toxic (rheumatic?) material within the body and thus in predisposed cases neuritis may result.

The infectious diseases, typhoid fever, diphtheria, tuberculosis, syphilis, etc., can cause wide-spread neuritis, as also can septic conditions (puerperium, etc.). The poison of gout, of rheumatism and of diabetes may cause a general neuritis — but with diabetes, syphilis and diphtheria, the neuritis may be localized.

In Japan a form of multiple neuritis has existed for centuries; it is there known as "kak-ke". Since the rice ration of the Japanese army has been supplanted by wheat flour and an addition of meat the disease has in great part disappeared from the army.

The natural inference would be that the disease is due directly or indirectly to faulty diet, but Dr. Albert A. Ashmead, of New York, who spent many years in Japan and who has investigated the disease, is firmly convinced that it is due to poisoning by carbonic oxide, and he reports a remarkable instance of the outbreak of the disease on board a ship sailing from the Philippine Islands bound for New York, and plentifully supplied with good food. Bad weather caused exposure and fatigue, and the cargo of unrefined sugar began to ferment giving off CO_2, so that the crew were evidently poisoned and the most virulent form of multiple neuritis attacked the ship's company causing several deaths. Dr. Ashmead's views have led the writer to insist in every case of neuritis upon a most abundant supply of fresh air. In tropical South America, and also in India, a similar type of the disease is known under the name beri-beri.

In these regions the affection occurs during the hot season, and the effort has been made to discover a special microbe as its originator.

The most frequent cause of multiple neuritis in the temperate zones is undoubtedly the use of alcohol, and in every case in which the evidences of the disease are present no effort should be spared to trace it to that cause. The writer has been so often surprised to find, on questioning servants and other attendants, that a continuous use of alcoholic beverages had existed unsuspected by the family physician, that he is now not satisfied in such a case to exclude alcohol as

the cause, unless the testimony against such view is overwhelming.

Other poisons introduced from without have undoubtedly caused multiple neuritis. In such cases the history, usually connected with some trade or occupation (mercury, bisulphide of carbon), or a "course" of drugging (arsenic, mercury,) point out the causal influence.

SYMPTOMS.—Injury to a nerve trunk, as by pressure, affects first the perineurium (perineuritis) then involves the epi- and endoneurium and intermediate connective tissue (interstitial neuritis) the usual evidences of inflammation, such as hyperæmia and cell proliferation being found on microscopical examination. From pressure the axis cylinder is injured and may be destroyed and then occurs secondary degeneration of the nerve fibre.

Primary or degenerative (parenchymatous) neuritis is of frequent occurrence, the neuritis beginning by degenerative changes in the axis cylinder, but the interstitial connective tissue soon becomes involved.

The most prominent symptom of an acute neuritis is pain, subjective, of the character already described and, objective, by pressure upon the nerve trunk and its ramifications. Paræsthesia and hyperæsthesia are common symptoms, while occasionally motor irritative phenomena, such as fibrillary contractions and cramps in the muscles supplied by the affected nerves are observed. Vaso-motor and trophic changes may be present; these are redness and swelling (œdematous),

herpes, "glossy" skin, bedsores, gangrene, pemphigus, etc., all in the distribution of the nerves inflamed. When the nerve is severely affected or the degeneration has advanced, hyperæsthesia gives place to anæsthesia or hypæsthesia, while paralysis and atrophy are to be expected.

The most common form of multiple neuritis seen in this country is that due to the long continued use of alcohol in even moderate doses. Hence it is more frequently seen in women than in men, since the latter seem more inclined to get intoxicated and then cease for a time the use of strong drink or else have delirium tremens. In my experience, however, I have seen multiple neuritis so often associated with delirium tremens in the very poor half-vagrant class having the alcohol habit that I am inclined to think that the distinction holds good only for those who are well nourished.

The onset of the trouble is really slow, although the outbreak of positive symptoms may be quite sudden. Vague pains and paræsthesias may exist for weeks with some weakness in the legs and many digestive troubles that mislead the family physician in the absence of any knowledge of the real cause. Then some morning on getting up and walking to her toilet table or bath-room the patient sinks to the floor rather than falls, and she is found to be paralyzed in the lower limbs. The muscles and nerve trunks are tender to pressure, the pains in the lower limbs increase and soon the upper extremities are involved,

although not to the same degree usually. The extensor muscles of leg and forearm are especially affected and the so-called foot-drop and wrist-drop are present. Examination may show areas of anæsthesia and lessened volume of the affected parts, while the reaction of degeneration determines the character of the atrophy when it is present. The anterior crural nerve being affected the knee jerk is of course gone and tremor of hands, lips, tongue and face may appear.

The functions of bladder and rectum are not much interfered with, but in my experience the stomach always or nearly always shows disturbance evidenced by thickly coated tongue and foul breath.

In estimating the influence of the infectious diseases in the production of a subsequent multiple neuritis, place should be given to the possible toxic effects of alcohol prescribed during the course of the former disorder, and this is especially true in typhoid fever and tuberculosis.

Whether the division of multiple neuritis into a motor, sensory and ataxic form is justified, is to the writer a doubtful point, since the predominantly ataxic or sensory forms are not at all frequent.

The metallic poisons, lead, arsenic, silver and mercury can cause multiple neuritis, and of these the first two more frequently than the others. In such cases the arms are affected first, usually, and more than the legs, the reverse being true of alcoholic neuritis. Lead seems to have a selective power in affecting the musculo-spiral nerve after the branch to the supinator

longus has been given off; but it also has a wide-
spread influence upon the structures of the central
nervous system, so that degenerative changes, especi-
ally in the anterior gray horns of the spinal cord and
also in the brain cortex, have been observed post-
mortem.

The local paralysis caused by the poison of diph-
theria is seen in the muscles of the palate, œsophagus
and larynx and even in the cardiac innervation. Just
how the paralysis of accommodation occurring frequently
after diphtheria, or of some of the external ocular
muscles, is produced, is difficult to say. In one case
the writer has seen a typical bulbar paralysis follow-
ing a severe sore throat which he believes to have
been diphtheritic. In another case a severe and
rapidly advancing multiple neuritis followed immedi-
ately the disappearance of paralysis of the palate mus-
cles occurring from diphtheria. It is to be kept in
mind that loss of the knee jerk is a common symptom
after diphtheria.

The association of neuritic (neuralgic?) symptoms
with loss of the knee jerk in diabetes is not as yet
understood. On the other hand the insufficiency of
Trommer's or Fehling's test for sugar in the urine is
now recognized and must formerly have led to errors
in diagnosis.*

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* Sugar. The sub-oxide of copper is precipitated by other substances
than sugar in the urine (uric acid, kreatin, kreatinin, etc.) and lately Salkowski
has called attention to the existence of a body termed pentose that occurs in
the urine in certain cases, which precipitates the sub-oxide from Fehling's
solution without boiling. The urine, so tested, shows in such cases fluorescence.
Such urine does not undergo fermentation by the addition of yeast; hence
the fermentation test should always be employed as a control test in all cases
of suspected diabetes or glycosuria.

The diagnosis of a neuritis in a peripheral spinal nerve is readily made out by recognition of pain, subjective and objective, in the affected nerve, by the loss or impairment of function, such as anæsthesia, paræsthesia, hypæsthesia, paralysis or paresis in the distribution of the nerve, and by the history of previous injury, exposure to cold, previous poisoning, alcoholic, metallic, etc., rheumatic (?), or of antecedent infectious or septic disease, etc. Erb's paralysis has been mistaken for poliomyelitis.

Multiple neuritis will be recognized by the paralytic and sensory symptoms in the extremities and limited chiefly to the distal half of each.

Where there is a considerable degree of ataxia and anæsthesia it may be mistaken for locomotor ataxia, especially when the onset has been slow. The absence, however, of bladder or rectal implication and of pupillary inaction and of a girdle sensation are against the existence of the latter, while the tenderness of nerve trunks and muscles, the paralysis or paresis of the limbs and the abnormal electrical reaction do not belong to tabes. Lightning pains as seen in locomotor ataxia do not belong to an ordinary neuritis, yet I have seen in more than one case the most frightful paroxysms of pain radiating from the back down the arms or around the trunk, these being evidently due to severe irritation of the nerve roots.

From poliomyelitis anterior the pains would be sufficient to differentiate a neuritis even in the presence of considerable atrophy, while the atrophy

itself is, in the two diseases, different in distribution;
in the former less in the distribution of nerve trunks
than in the muscles associated in some one definite
action.

In myelitis transversa there are no spontaneous
pains unless the nerve roots are implicated; there
is no tenderness of nerve trunks, no atrophy of
muscles, while there is exaggeration of deep re-
flexes, a thing not present in neuritis except in the
very earliest stage. In myelitis lumbalis, leading to
atrophic paralysis and loss of knee-jerk, paralysis or
paresis of bladder and rectum is to be expected.

The prognosis in multiple neuritis is good, pro-
vided the cause has been removed, or in cases of
paralysis following the severing of a nerve if the
cut ends be in apposition, the connective tissue sheath
of the cut ends serving as a guide or pathway for
the new growth from the proximal uninjured part.

Multiple neuritis has a tendency to recover, except
in the pernicious forms, in which there is some im-
plication of the cardiac nerve supply. In the alco-
holic form when delirium tremens is present the
prognosis is grave as long as the latter condition
remains, or if pneumonia develop. Cases of multiple
neuritis without the complications recover in a few
months, or after many, according to the degree of
severity and consequent amount of degeneration.

Concerning the homeopathic treatment of neuritis,
the general considerations as to treatment already
given should be borne in mind. The causal indi-

cation is always of the first importance in select-
ing a remedy. Hence, in cases in which trauma
(pressure, etc.) has produced a neuritis, *Arnica* is,
in the writer's experience, the remedy first to be
thought of, and should be given in a moderate po-
tency and not frequently repeated. *Hypericum* has
received great praise for its influence in restoring
a nerve after traumatic influences, but, in the
writer's opinion, it has less influence than *Arnica*,
possibly because it seems to have a selective in-
fluence upon either the terminal organs of the sens-
ory fibres, or on the posterior nerve roots as they
enter the spinal cord. In several instances, where
the only clinical presumption possible was special
implication of the posterior nerve-roots, the remedy
in third dilution acted admirably in relieving pain
and bringing about a rapid cure, and in at least
one case in which an ascending neuritis of the
arm followed a punctured wound of a finger-tip
this remedy in 200th seemed to be the only one
that made the patient comfortable, even after mor-
phine had been used in vain for this purpose.

Other remedies, such as *Ruta graveolens*, *Rhus toxi-
codendron*, *Dulcamara*, *Ledum palustre*, or *Apis melli-
fica* will be thought of should their special causal
indications be present.

When the use of alcohol can be fairly charged as the
cause of a neuritis, even of severe type, there is no
one remedy (unless special contra-indications are pres-
ent) so valuable as *Cimicifuga racemosa* given in 3d or

6th; in a local neuritis whose immediate cause is trauma, while the predisposing cause has been the use of alcohol, *Arnica* had better be given first, to be followed later by *Cimicifuga racemosa*. Next in rank to *Cimicifuga racemosa* are *Ranunculus bulbosus*, *China officinalis* and *Bryonia alba*, and in more chronic cases *Arsenicum* and *Lachesis*. *Ledum palustre* ought to be a valuable remedy in alcoholic neuritis, but the writer has not used it in the multiple affection, owing possibly to the limitations of the direction of the pains produced by it.

In other forms (non-alcoholic) of neuritis remedies must be chosen by the symptoms, but here again some cause, such as exposure to cold or wet, will lead the prescriber by a short-cut to the selection of the remedy. *Rhus toxicodendron*, *Ruta graveolens*, *Calcarea carbonica*, *Dulcamara*, *Natrum sulphuricum*, *Bellis perennis* and many others are to be studied for this purpose.

A slight trauma may set up a neuritis in a patient whose nutrition is below par from causes other than the use of alcohol. Quite recently a patient called on the writer suffering from neuritis of the median and ulnar nerves of the left arm, caused directly by a woman in confinement having grasped the arm with great force during one of her pains. There was intense subjective pain, with paresis of the flexors of the forearm and hand. A remedy was given with some slight relief, and on the next visit the doctor-patient stated, as an odd fact, that for several years

previous there had been a chronic diarrhœa which no remedy seemed to reach and that it had ceased since the existence of the neuritis. Further investigation showed that the diarrhœa had been caused by plunging into the sea while overheated, the water being very cold at the time. The diarrhœa, which had stopped during the neuritic attack, returned as soon as the neuritis was lessened by the remedy. Acting upon this hint, *Bellis perennis* was given in the belief that the diarrhœa must be cured first before the neuritis would go. The result more than justified this opinion, for both diarrhœa and neuritis disappeared, the latter, however, not at once, for a relapse was caused by clapping the hands at a public entertainment. Two doses of *Arnica radix*, followed by a few of *Bellis perennis*, restored the patient, whose arm, however, had to be humored for several weeks.

At times the location of a neuritis will enable the physician to select the remedy. For instance, any neuritis in the distribution of the nerves from the lumbar and sacral plexuses seems readily influenced by *Berberis vulgaris*. If it is limited to the lesser sciatic, *Æsculus hippocastanum* seems to be better indicated, and if in the anterior crural, *Pareira brava*. The rectal symptoms of *Æsculus hippocastanum*, or the bladder symptoms of *Berberis vulgaris* or of *Pareira brava*, are strong indications, even though they themselves have long preceded the neuritis.

So a deltoid paralysis (circumflex nerve) of months' standing, occurring in a farmer, and presumably from

cold, has been relieved in a few days by *Sanguinaria Canadensis.* For a root neuritis, or trouble simulating this in the upper dorsal roots, *Anantherum muricatum* 30 has acted in a marvellous manner in several cases.

Of the large number of substances already mentioned as causing multiple neuritis *Arsenic, Mercury, Lead, Bisulphide of carbon,* and *Copper* are also well-proven homeopathic remedies. *Carbon monoxide* has a place in the homeopathic materia medica under the title *Carboneum oxygenisatum,* but there are many drugs whose provings show symptoms similar to those of multiple neuritis. But we are not limited to remedies known to cause inflammation of peripheral nerves, and should hold in mind the characteristic symptoms, modalities, and especially the causal ones as our guides.

The writer has known *Phosphorus* 6 to make a rapid cure of post-diphtheritic multiple neuritis, the selection of the remedy being made on the ascending of sensory and motor paralysis from the ends of fingers and toes. The diphtheria toxine* (not anti-toxine), in the 200th potency, rapidly cured a hospital case unable to walk from post-diphtheritic polyneuritis.

As to the care of a case of multiple neuritis, the cause, if found, having been removed, warmth and good nourishing food, with an addition of assimilable fat, are the chief things. In applying

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*Obtained through the kindness of Dr. Paul Gibier, Director of the Pasteur Institute, New York, and potentized for the writer by Dr. Martin Deschere.

hot bottles or hot-water bags to the affected limbs,
the danger of burning must be kept in mind, for
the anæsthesia or hypæsthesia may prevent the pa-
tient from appreciating the degree of heat used,
and the tissues during the existence of the disease
have a much lowered vitality. Troublesome ulcers
may thus be caused.

Motion must be avoided if it produce much sub-
jective pain. Massage is, in the writer's opinion,
injurious in any stage of the affection. Galvanism
is often (not always) of marked value, but should
not be employed, as a rule, until the acute pains
have somewhat subsided; occasionally the anode to
the site of great pain, the cathode in some indif-
ferent part of the body gives great temporary re-
lief, but at times this is for a short while only,
and may be followed by marked and long-continued
aggravation. The benefit from the use of galvanism
is most in the later stages, when spontaneous pain
has greatly lessened; the application should be
cathode stabile at an indifferent part, anode labile
along the affected nerve trunk, small currents of 2
or 3 m-a for 2 or 3 minutes to each limb. In old
cases where atrophy is markedly present, the same
electrical treatment as that followed in poliomyelitis
anterior should be used. When contractures exist,
manipulation and extension of the contractured muscles
will often overcome the deformity and bring about
restoration of structure and function.

Neuralgia.

The word neuralgia means nerve-pain, but it is applied only to pain existing in a nerve without any anatomical change in the latter.

The pain in neuralgia is of different qualities; it may be boring, tearing, crushing, lancinating, etc. It generally comes in a succession of outbreaks, each lasting a few minutes or, at times, much longer. At the end of the paroxysm there may be entire relief until a new one begins, but often there is during the interval a considerable substratum, so to say, of pain that is continuous. When all pain ceases, not to return, the attack is over.

An attack of neuralgia may be brought on by different causes, exposure to cold being the chief one; but mental excitement, excessive fatigue, error in diet, etc., are, at times, provocative.

The subjects of neuralgia are neurotic or debilitated, or have gouty or rheumatic constitution. The affection first appears, in the majority of cases, in early adult life; it rarely occurs before the period of puberty.

Neuralgias may be divided into idiopathic, reflex and symptomatic forms, according to causation. When due to irritation of the nerve directly, as by a tumor within the skull, irritation set up by a decayed tooth, etc., it is symptomatic; when caused by disease of some distant organ it is reflex, and when no cause is assign-

able other than a general state of the system affecting the nerve, it is idiopathic.

The pathology of the affection is in doubt. It is easy to assume that the affection must specially involve the *nervi nervorum*, but the irradiation or streaming out of the pain apparently to other nerve trunks is best explained by locating the real seat of the trouble in either the posterior spinal ganglia or in the cells of the posterior gray horns of the cord, and extension of irritation to other cells will send up to the cortex impulses of pain felt as coming from the related peripheral nerve. Reflex neuralgias are produced in a similar way and muscular spasm in the part may be aroused by irritation sent to the anterior motor cells. Gowers gives an illustration showing a focus of softening in the pons, involving part of the sensory nucleus of the fifth nerve; during life it had caused severe neuralgic pain in the face.

Hyperalgesia (to a light touch) of the skin covering the affected region is observed in many cases, but often firm pressure will ameliorate the pain. Tender points may be found in the course of the affected nerve, at its emergence from a bony foramen or in its course over a bone or where it passes through fascia.

The diagnosis of neuralgia is made from the location of the pain in one nerve which changes place from moment to moment, and from the absence of evident organic change in the nerve (paralysis in a mixed nerve, anæsthesia); in neuritis pressure anywhere along

the affected nerve is painful, while in neuralgia if firm pressure be made it often suppresses the pain, as above stated.

The occurrence of pain in paroxysms, with freedom in the intervals between the attacks, points to idiopathic neuralgia rather than to organic disease affecting the nerve. In reflex neuralgia the distant cause may be difficult to discover.

Long continuance of neuralgia may result in setting up some amount of organic change in the nerve. Eruptions such as herpes, pemphigus, etc., in connection with pain in a nerve are evidence of organic change in the nerve itself or its spinal ganglion, or, in case of the fifth nerve, in the Gasserian ganglion.

Neuralgia of the Fifth Nerve.—Trigeminal Neuralgia. —Prosopalgia.—Tic Douloureux.

This is the most common form of neuralgia, indeed it is more common than all other forms taken together. It is the most typical in the characteristics of pain, paroxysms, free intervals, etc.

It may attack any one of the divisions of the nerve or two of them or all three.

When the first division is the seat of the trouble the pain is generally over the eyebrow; it may extend down the side of the nose or be felt in the eye. Painful pressure points are just above the supra-orbital foramen, on the nose near the inner canthus and on the bone just above the outer canthus.

When the second division of the nerve is affected,

pressure points may be found at the infraorbital fora-
men, at the side of the nose and on the malar bone.
When the third division is involved, the points are
in front of the tragus or somewhat above this, at the
parietal eminence and at the site of the mental fora-
men.

The eyeball itself may be a pressure point when
its supply from the first branch of the fifth is involved;
or an ocular neuralgia may exist alone.

In elderly persons neuralgia alveolaris in the tooth-
less gums has been described by several observers;
the writer has seen loosening of a tooth (from absorp-
tion of the alveolar process) follow an attack of facial
neuralgia, not once, but many times in the same indi-
vidual. The loosened tooth, if treated and kept,
seemed to receive the brunt of such trophic change
in subsequent attacks, but when it had finally to be
drawn, another attack would find a fresh victim in
a hitherto firm tooth.

The pain in an attack of trigeminal neuralgia varies
greatly, but in the paroxysms there is a "shooting"
character with a "cutting" quality. At times it is
a crushing of the most intense kind; at others as if
the tissues were rapidly torn through as one might
tear a strip of cotton cloth. The peculiarity of the
affection is its irradiation from one branch of the nerve
to another; the supraorbital region may be intensely
painful, but in a few moments there may be a streak
of pain in the lower jaw or a long stab beside the
nose or just in front of the ear.

Or the pain may play along the face as the so-called "heat" lightning plays across the nocturnal sky —here the lancinating quality of the pain is not marked, but is replaced by a feeling as if the nerve were rapidly crushed. When this lambent characteristic is present the writer has observed a chilled or cold sensation in the nerves, and as a very severe attack with these peculiarities was accompanied in part of its course by an outbreak of herpes, he is of the opinion that such attacks show irritative or even inflammatory changes in the Gasserian ganglion.

During a paroxysm of pain the sufferer can bear no external irritation—every thing is unbearable—to move the head or body, to open the mouth, to be touched near the areas of pain, all give increase of agony. A draft of air, an increase of light, a sudden noise, is intolerable, hence the myriad of suggestions to use hot applications or cold ones, to do this or that, have perhaps some beneficial effect in distracting the attention of the now irritable brain, become so from the intensity of the suffering.

The attack may last hours or days and then gradually disappear; or, happily, it may be cut short under the influence of a well-chosen remedy.

One form of trigeminal neuralgia termed by Trousseau **epileptiform neuralgia** (a most unsuitable title) deserves attention. In these cases the sufferer is attacked with a suddenness that is appalling by an outbreak of pain in one of the branches of the fifth nerve. The suffering is of the most intense character

and it may last a minute or two. In the agony of
it the patient's face is drawn up, his hand goes in-
voluntarily to the seat of pain and he remains bent
over and immovable until it ceases. Or, unable to
control himself, he literally runs about the room, as
if out of his senses, or presses the painful part with
his hand or against any object. The paroxysm being
over, the patient is himself again, but a sudden motion,
even in answering a question, may bring on a return
of the pain—and indeed such patients are always in
terror. The writer has studied out remedy after
remedy for this trouble, but so far has failed to affect
it. The patients having this affliction are usually in
the latter half of life, but apart from their special
trouble appear to be in good health. Among the old-
school physicians the disease is considered incurable.

The treatment of prosopalgia by homœopathic rem-
edies is one of the most satisfactory experiences in
the whole domain of neurology. Of course any rem-
edy that is specially indicated must under the circum-
stances be prescribed, yet it so frequently happens
that certain drugs especially influence certain nerves
that they may almost be considered specifics. The
side affected, the branch of the nerve, the causal
conditions, the conditions of aggravation or amelioration,
the underlying reflex causes, must always be taken
into account and a study made thereon. *Spigelia,
Kalmia, Gelsemium, Magnesia phosphorica, Belladonna,
Bryonia, Rhus toxicodendron, Colocynth, Natrum muria-
ticum,* and *Nux vomica,* have in the writer's hands been

of remarkable efficacy. *Natrum sulphuricum* 6, (given
because of the special yellow-green coating of the
tongue, which led to further study of the case and
brought out the history that the trouble began while
living in a very damp house) cured a case that had
lasted for years, and which came to the writer for
opinion as to the advisability of neurectomy. *Mer-
curius* has often been of service, and more than
one case has been cured by removal of amalgam
fillings in the teeth, or by changing a red-rubber
dental plate (colored with cinnabar) to a gold one.

The dentists refuse to believe that amalgam fill-
ings or red rubber plates can give to the saliva
any amount of mercury, forgetting or not knowing
that the saliva at times contains sulpho-cyanide of
potassium, which can readily decompose in the
presence of mercury, and form a mercurial sulpho-
cyanide. The sulpho-cyanide is not always present,
indeed may only appear in morbid conditions; *vide*
Hoppe-Seyler's Physiologische Chemie.

Cervico-occipital neuralgia. A not common form.
The pain is as in pure neuralgia, is in the four
upper cervical nerves, and is chiefly in the distri-
bution of the great occipital nerve, in which region
it is not infrequently bi-lateral. It is less par-
oxysmal, and the exacerbations are less severe than in
trigeminal neuralgia. It may extend down the neck
from the jaw, involving the small occipital nerve.

Cervico-brachial neuralgia is a rare form. The
pain is in the nerve supply from the four lower

cervical and the first dorsal nerves. It may affect
any part of hand or lower arm and posterior part
of the neck; it is greatest in the axilla and in
the ulnar nerve. Painful pressure points may be
found in the axilla, at the posterior border of the
deltoid and on the ulnar nerve behind the elbow,
and on the front of the wrist. There is more or less
constant pain, with very acute paroxysms, often
brought on by moving the arm, or by exposure to
cold. It is stated to result from injury, but such
a history ought to lead to examination for inde-
pendent or associated neuritis of the plexus.

The dorsal nerves are mixed nerves; each di-
vides into a large anterior branch and a small pos-
terior one, the first dorsal contributing largely to the
brachial plexus, its anterior or intercostal portion
being small. The first six of the anterior branches
are intercostal nerves, and are exclusively distributed
to the chest and its coverings; the last six supply
the walls of the chest and abdomen.

Intercostal neuralgia is an affection frequently
seen, generally in women who are debilitated or neur-
asthenic. The nerves oftenest affected by neuralgia
are from the fourth to the eighth. Painful pressure
points are at the side of the related vertebral
foramina, in the axillary line and near the sternum.
The pain is more or less continuous, but has severe
exacerbations, sharp or stabbing in character, felt in
the course of the nerves. The left side is most
frequently affected, but both sides at times are

involved. Exposure to cold is a common exciting cause; the neuralgia may be produced symptomatically by disease of the vertebræ or ribs, by growths within the spinal canal, etc., and by external injury to the chest wall.

It is to be distinguished from pleurodynia, which is a pain located in one spot, and not following the course of an intercostal nerve; there is great tenderness on pressure. The pain in pleurodynia is aggravated by respiratory movements of the chest, and is probably due to a neuralgia of the pleural nerves (Gowers).

Neuralgia mammalis. Mastodynia. Pain in the breast may be idiopathic or symptomatic (tumor). It occurs mostly in women, but has been observed in the male. It affects one gland only, but may involve the lateral as well as the anterior branches of the related intercostal nerves. Terrillon has observed it in women, either stout or slender, whose dependent breasts have not been supported, thus by interference with the local circulation directly causing the affection. The same writer considers ill-fitting corsets to be a cause of the trouble. The paroxysm of pain may last but a few minutes, or may extend over hours; it may spread to the back, the axilla, the inner side of the arm, or even to the fingers; at times it is of great intensity. A painful pressure point has been observed below and to the inside of the nipple or at the upper outer edge of the gland. The nipple itself may be extremely tender to touch. Mastodynia

occurs after puberty, during pregnancy, or during lactation, and may give rise to much apprehension through fear of cancer.

Lumbo-abdominal neuralgia involves the lumbar nerves or their derivatives. It is seen in women chiefly, and in the middle period of life. Little is known of its etiology, but cold, exposure and intrapelvic disorders irritating the cords of the lumbar plexus, are among the causes ascribed.

The pains, dull and continuous, become sharp in the paroxysms, are located in the lower back, and stream down the buttock or on the upper edge of the pelvis, or to the hypogastrium, inguinal region and the genitals, on one side. They may involve the inner or outer side of the thigh.

Painful pressure points may be found outside of the first lumbar vertebra, above the crest of the ilium, over the middle of Poupart's ligament, or even at the lower surface of the labium majus (or scrotum).

Anterior crural pain is not a rarity; it has been observed in connection with bladder troubles and in conditions of so-called lithæmia.

Of neuralgias in the nerves of the sacral plexus the chief one is **sciatica**. It is nearly as frequent as trigeminal neuralgia, occurs in men four times as often as in women, and in the majority of cases begins between the ages of 30 and 50. It rarely begins before 20.

The predominance of males among the sufferers from sciatica is ascribed to their special conditions

of life, such as exposure to cold, wet, over-exertion, etc. Living in damp habitations, sleeping in wet clothing or on damp ground, working in water, sudden cooling while over-heated, have repeatedly caused an outbreak of the disease.

Traumatism plays also a *rôle*. Falls upon the buttocks, pressure of a tumor within the pelvis, of the gravid uterus, of the forceps during labor, even large accumulation of fæces in the rectum, have been assigned as causes. Direct pressure, long continued, as in sitting on a hard bench, has been a cause.

Sciatica may be symptomatic of vertebral disease, tumors within the spinal canal, meningitis, etc., but in such cases it is more apt to appear bi-laterally. It is not infrequently an accompaniment of diabetes and often the condition of pain varies directly with the increase or decrease of sugar in the urine.

The pain may begin suddenly, but often for a day or two preceding there is discomfort about the hip and thigh on movement. It is an aching with paroxysms of lancinating or tearing, extending at times downward to the foot or upward to the sacral region; in most cases the pain is in the back of the thigh and in the calf, but it may be chiefly in the anterior part of the leg or in the foot.

Painful pressure points may be found at the sciatic notch, between the trochanter major and tuber ischii, in the middle of the thigh, in the popliteal space, along the fibula, in the middle of the calf, behind the external malleolus and on the back of the foot.

The lesser sciatic may be involved and then there will be superficial tenderness on the back of the thigh and extension of the pain to the sacral region.

The pain is usually increased by motion and by pressure and often the patients assume peculiar positions when sitting or standing. At times the pelvis is raised on the affected side and the spine curved toward the well side; at times both conditions are reversed. Various explanations of the scoliosis thus produced have been made, paresis of the erectores spinæ, spasm of the same, and implication of the nerves of the lumbar plexus, being among the latest.

The disease may last a few weeks or may continue, with periods of remission, during many months.

Pathology. Gowers considers that most cases of primary sciatica are cases of neuritis. Some wasting of muscles occurs and changes in their electrical reactions are found at times—conditions belonging to neuritis and not to neuralgia. Changes in the nerve sheath, at times extending to the interstitial tissue, have been found in most of the cases examined. The neuritis is thus a perineuritis.

The diagnosis of sciatica consists chiefly in excluding any distant cause of the pain. Such causes have been mentioned on a preceding page. Between a sciatic neuritis and a pure neuralgia of the nerve, the tenderness of the nerve trunk, the subjective pain in it and the wasting of muscle are in favor of the former; the pain in branches of the nerve rather

than its trunk and a history of previous attacks of neuralgia elsewhere, are in favor of the latter.

The prognosis is good as to cure in cases of sciatica not due to symptomatic causes mentioned on page 78. In such cases the sciatica is only curable by removal of the cause. Homœopathy has had many and brilliant successes in the treatment of the disease.

Coccygodynia is a neuralgia of the last sacral and the coccygeal nerve, the pain being chiefly felt at the tip and on the posterior aspect of the coccyx. It occurs oftenest in women and results from dislocation of the coccyx during labor, from a fall on the buttocks or similar injury, and from disease in the region itself. It is seen chiefly in hysterical or neurasthenic persons. It is said to arise spontaneously, but this is rare.

Treatment of neuralgia is a matter of studying out the remedy. Some of the remedies for trigeminal neuralgia have been mentioned on page 73; to mention all the possible remedies would be to recapitulate the names of a majority of our drugs. Experience, however, has shown that many cases present repetitions of indications for certain drugs, so that the list of those most often found serviceable in trigeminal neuralgia becomes short relatively. In recent cases *Aconite* is indicated when its special symptoms are present, especially restlessness and anguish, and when exposure to sharp cold has been the cause. *Belladonna;* the pain shoots to different parts, there is redness of the face and aggravations from light,

noise, touch (but hard pressure may relieve), a jar, or draft of air. Other remedies that have brought rapid relief at the writer's hands are *Kalmia, Spigelia, Plantago major* (pains sharp and "play" chiefly between the teeth and the ear), *Magnesia phosphorica, Nux vomica, Menyanthes* (relief by hard pressure with the hand), *Mercurius* and *Cimicifuga.*

In patients who are subject to trigeminal neuralgia *Arsenicum, Argentum nitricum, Phosphorus, Platinum, Stannum or Staphisagria* will frequently be found indicated. Malarial cases may require *Natrum muriaticum,* especially after abuse of *Quinine,* while *Cedron* and *Quinia sulphate* among their symptoms have marked periodicity in the return of pain.

In ciliary or ocular neuralgia *Allium cepa, Paris quadrifolia, Croton tiglium* have repeatedly been of value, while *Gelsemium, Belladonna, Nux vomica* and *Mezereum* are highly recommended.

In occipital neuralgia *Cimicifuga, Picric acid, Ammonium picricum* and *Carbolic acid;* in brachial and intercostal neuralgia *Aconite, Spigelia, Anantherum muricatum* 30, *Chelidonium* (right side) and *Ranunculus bulbosus* (region of upper ribs), *Bryonia, Kali carbonicum* and *Rumex crispus* (region of lower ribs, left side) have been efficacious.

In mastodynia *Belladonna, Chamomilla, Conium, Croton tiglium, Helonias, Cimicifuga* and *Phytolacca* have been recommended.

Under the heading "sciatica," Knerr in the repertory to Hering's Guiding Symptoms gives seventy-three

remedies, most of which are marked with the sign of cure. They embrace practically all the remedies already mentioned as indicated in neuralgia. In the writer's hands *Gnaphalium* has acted curatively in a remarkable way more than once, and if unable to obtain satisfactory indications for some other drug, he would choose, as the remedy for sciatica, *Gnaphalium*. Good results have followed the use of *Colocynth*, *Rhus toxicodendron* and *Ammonium muriaticum*.

Repetitions of sciatica wear upon the patient's strength and gradually bring about gastric, intestinal and circulatory disturbances, which in turn react unfavorably upon the affected nerves. In severe cases of this kind the great constitutional remedies will do more in the direction of giving lasting improvement than remedies chosen simply on the local symptoms.

Coccygodynia is so often associated with other pains in nervous or hysterical women that its symptoms must be taken with others in prescribing. When the trouble is alone, the causal indication of trauma (bruise or dislocation) will call for *Arnica*, or *Rhus*, or *Ruta*. *Kali bichromicum*, *Cistus*, *Antimonium tartaricum*, *Paris quadrifolia* and *Petroleum* have with other remedies been reported as valuable in the treatment of the affection.

In case of failure of remedies to help, manipulation of the coccyx through the rectum or vagina may undo a displacement, or removal of the bone may be resorted to, a procedure that has been followed in some cases by success, in others by failure.

Morton's neuralgia, or **metatarsal neuralgia,** is located at the metatarso-phalangeal joint of the fourth toe, usually. It is of traumatic origin. Lateral pressure upon the joint structures by a narrow shoe, and consequent forcing the end of the fifth metatarsal bone against the fourth, compressing an intercurrent branch of the plantar nerve, or treading through a thin sole upon a stone, with direct injury to the part, are common causes. Some amount of dislocation of the metatarsal bone undoubtedly exists in many cases.

Cure is to be obtained by removal of the cause— that is, by wearing thick-soled, roomy and well-fitting shoes; roomy and well-fitting stockings are equally essential.

During an attack relief will be found by manipulating the last three toes by drawing them forward and downward and reversing the procedure; the patient will soon find out which brings the desired relief. When at home the patient should wear as much as possible roomy slippers having felt insoles.

When once well-established, the affection is, even after disappearance by removal of the cause, liable to return from too long standing, or taking too long a walk.

Remedies for recent cases are *Arnica, Hypericum, Rhus toxicodendron* and *Ruta;* for cases of long existence, besides these, *Sabadilla* and *Cannabis Indica.*

Erythromelalgia is the name given to a special affection of the extremities, usually the feet, in which

the feet (especially the soles) are red, somewhat
swollen and extremely painful, both when at rest and
in motion. In the cases seen by the writer there was
a history of an excessively long walk under conditions
of fatigue or of other depressing influences. At first
the trouble may appear in attacks, but soon it be-
comes more or less continuous, is often worse at night
and on walking, and is better in cold weather. Lewin
and Benda, after a study of all the cases reported,
have come to the conclusion that it is not a disease
sui generis, but that in some of the cases it is a
special form of neuritis, in others it is part of a
general neurosis, and in still others it is an accom-
paniment of brain or cord affection.

The cases seen by the writer have been apparently
neuritic. One case was cured by *Sabadilla;* the
others did not remain under treatment. The most
promising remedies are *Sabadilla, Phosphorus* and *Can-
nabis Indica.* *Squilla* has the symptom "soles red
and sore when walking."

Acroparæsthesia. This term Schultze, of Bonn,
applies to an affection long known to neurologists
and described commonly as "numb fingers." The
trouble seems to be a distinct disorder and is seen
most often in those whose work compels them
to have the hands in water frequently. It is seen in
washerwomen, in dishwashers in hotels, and one case
of the writer's was a man who handled ice con-
stantly. There is undoubtedly impaired nutrition as
an underlying condition in all cases, and in many

the use of beer. The symptoms are usually prickling or "pins and needles" sensation, affecting the ends of the fingers, sometimes extending up into the hand and sometimes limited to the ulnar or the median distribution. A similar condition has been described as affecting the feet. The symptoms are worse at night and from exposure to cold. The disease is very intractable in many instances.

There is no known pathology, the disorder being held by different observers as a neurosis, an acroneuritis or as a vaso-motor affection, the latter because in some instances there is either shrivelling of the ends of the fingers or slight alteration in color.

Treatment should be directed by the general symptoms, the special symptoms not often leading to the selection of any efficient drug. Electricity, especially faradism, is helpful in lessening the annoying prickling, but galvanism seems to be more potent in the direction of cure.

Among our remedies, however, a not inconsiderable number have caused numbness of the fingers; chief among these are *Phosphorus* especially, *Kali carbonicum*, *Lachesis*, *Staphisagria*, *Crocus sativa*, *Nux vomica* and *Silicea*.

The Spinal Cord.

The spinal cord is that portion of the central nervous system placed within the spinal canal. It extends from the level of the upper border of the axis to that of the lower border of the first lumbar vertebra in the adult; in the new-born infant it reaches to the lower border of the third lumbar.

Around the central canal, which is all that remains of the cavity of the original neural tube, the gray matter of the cord is located in such manner that its cross-section has something of the appearance of the letter H. The white matter is outside the gray and is composed of medullated nerve fibres. The cord is incompletely divided into two lateral halves by an anterior fissure and a posterior septum of glia tissue.

By reference to the illustration the location of the different fibre-tracts, cells, etc., can be readily seen. In the anterior limbs of the gray matter are placed several groups of large ganglion cells whose axis cylinders gathering together at successive levels form the anterior or motor nerve roots. Similarly, from the cells of the posterior spinal ganglia, the nerve fibres form at successive levels the posterior or sensory nerve roots, which enter the cord at the extremity of the posterior horn, or in the column of Burdach; the latter divide into ascending branches which pass upward,
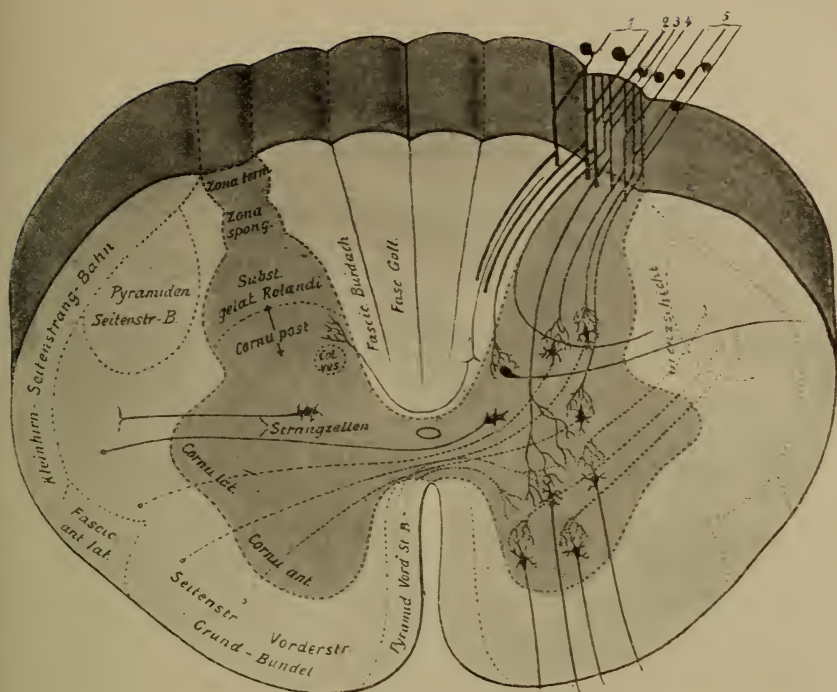
Fig. 14.

Fig. 14, from Edinger, giving general idea of the structure of the spinal cord. A segment of the spinal cord is shown, the posterior spinal nerve roots of the segment represented (diagrammatically) as arising from single cells (of the posterior spinal ganglion), the centrally directed ones enter the cord, some directly into the column of Burdach, some into the gray posterior horn. The fibres or collaterals that end at or near the level of entrance are seen to do so, as a brush-work surrounding cells in the gray matter from which new fibres arise; fibres which are to ascend higher than the level of entrance appear as if cut off.

The anterior spinal nerve roots are seen to arise from the ganglion cells in the anterior gray horns; near one motor cell, a fibre from the posterior root is seen, ending as a brush work, thus completing the reflex arc or reflex mechanism.

In the gray matter the anterior, lateral and posterior cornua or horns are sufficiently well designated; Col. ves.=vesicular column of Clarke. In the right half of the picture a fibre from the posterior root is seen ending in a brush work about a cell of Clarke's column, from which a new fibre arises and passes to the extreme lateral edge of the cord, there to turn upwards as part of the direct cerebellar column of white matter. The gelatinous substance of Rolando, the zona spongiosa and the zona terminalis are plainly indicated.

In the white matter Pyramid. vord. St. B. and Pyramiden Seitenstr. B.=the anterior or direct and the lateral or crossed pyramidal tracts, respectively.

Kleinhirn-Seitenstrang-Bahn=direct cerebellar column; Fascic. ant. lat.=antero-lateral or Gowers' column; Seitenstr. and Vorderstr.=lateral tract and anterior tract respectively; Grund-Bundel =ground bundle or basis bundle, and Grenzschicht=limiting layer, both of short fibres probably; the columns or fasciculi of Burdach and Goll are indicated by their names.

giving off on the way collaterals, and end in a brush work in the neighborhood of cells at the top of the cord, and descending branches which passing forward at the level of entrance descend some distance and end in bushlike expansions near the cells of the anterior gray horn

of one or both halves (forming a segment of a reflex arc) or about the cells of the column of Clarke.

The fibres of the column of Burdach are displaced toward the median line by new fibres entering at higher levels, thus forming the column of Goll.
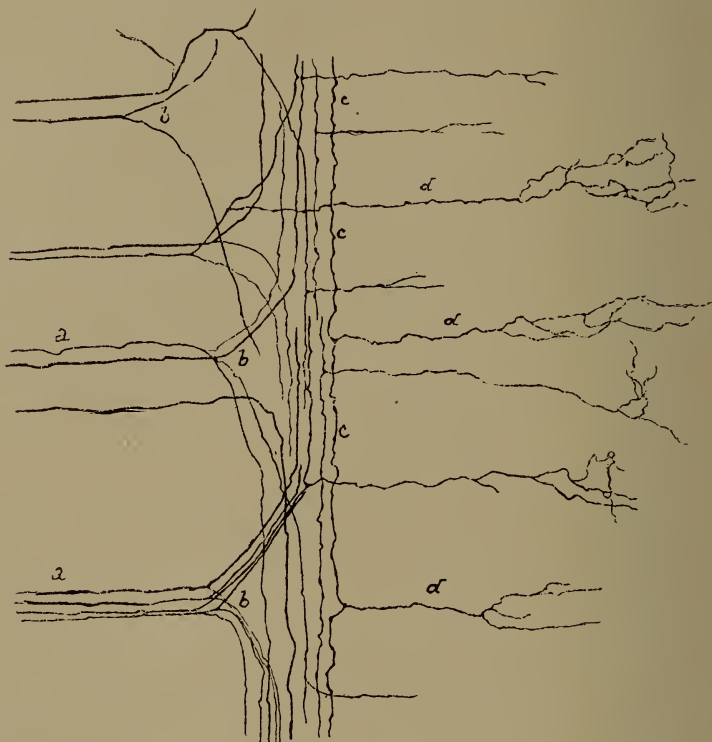

Fig. 15.

Fig. 15.—Course of some fibres and their collaterals.   From an antero-posterior longitudinal section of the spinal cord of a human embryo 20 centimetres in length.   a=fibres from the median division of a posterior root ; b=their bifurcations in the most anterior part of the gelatinous substance of Rolando ; c=longitudinal fibres of Burdach's column ; d= collaterals.   From Lenhossek.

The fibres entering the gray matter have a short course; they end in brush-work near cells in the gray, from which new fibres arise and pass upward in the columns indicated in the illustration.

The long fibres (primary neurones) of the columns

of Goll and Burdach respectively, are for the trans-
mission of muscular sense impulses; those of the
direct cerebellar tract are possibly for transmission
of similar impulses, but may have to do with impulses
from the organs of vegetative function, since the vesi-
cular column of Clarke begins (and consequently the
direct cerebellar tract) at the level of the lowest
pair of sympathetic ganglia ; the fibres of the antero-
lateral column carry up, probably, the sensory im-
pulses of pain and temperature. The exact route of
the impulses of ordinary tactile sensibility may be by
a part of the postero-external column, but may be
in part through the anterior ground bundles.

The paths for voluntary motor impulses from the
cerebrum are in the pyramidal tracts, the anterior or
direct pyramidal tract coming from the cerebral hemis-
phere of its own side, the lateral or crossed pyramidal
tract from that of the opposite cerebral hemisphere.
The fibres in each case on reaching the destined
level, turn into the anterior gray horn of the same
side and end in brush work about the large ganglion
cells there, from which the fibres of the anterior
(motor) nerve roots arise. The view that the fibres
of the direct pyramidal tracts cross to the opposite
anterior horn through the anterior commissure is now
denied. (Lenhossek.)

The existence of collaterals from the fibres of the
column of Goll has not been observed by Lenhossek.

Short fibre tracts occupy the white areas of the
cord as yet unmentioned, i. e., the residues of the

anterior and lateral columns. These, in part, carry fibres connecting different segments of the cord or opposite halves of the cord (via anterior commissure).

The mechanisms in the cord for reflex actions of different kinds are centres with afferent and efferent connections. Their positions are given on page 100.

The nerve cells in the spinal cord are many, and can be divided into groups. They are in the gray.

The large ganglion cells of the anterior gray horns have a very peculiar structure, as shown in Figure 16a. They are star-shaped or multipolar. Their processes divide and subdivide at a little distance

Fig. 16 a.

Fig. 16a.—Large motor ganglion cells from anterior gray horn of an ox. The axis cylinder process or neuraxon is downward. The other processes are dendrites. Only the beginnings of the processes are shown. The complicated details of the cell body are shown here ; in Fig. 16b the processes are shown at great length, but the Golgi stain there used leaves the details of the cell body undistinguished. Highly magnified.
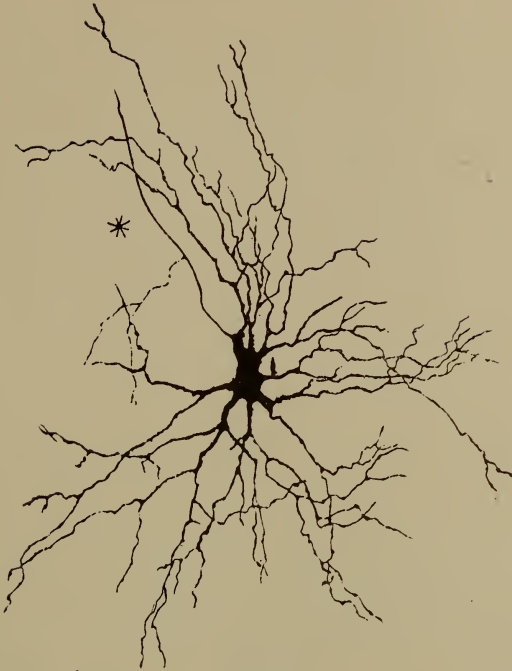
Fig. 16 b.

Fig. 16b.—Motor or ganglion cell from the anterior gray horn of the spinal cord of a human fœtus, 30 centimetres in length. A collateral branch (Golgi) from the axis cylinder or neuraxon is seen ; near the latter the star is placed. From Lenhossek. Highly magnified.

from the cell and end in arborization. Their axis cylinder process passes forward as an anterior or motor nerve root.

The cells are in groups, each group being in relation to a special muscular function or class of muscles.

In the middle and lateral areas of the gray matter are placed many cells, which give rise to fibres that pass outward and turn up in the lateral and anterolateral white columns.

At the junction of the posterior horn with the middle portion of the gray is a column of cells known as the vesicular column of Clarke. The cells

send out fibres that pass laterally to the edge of the cord, and passing up form the direct cerebellar tract, posterior to the antero-lateral tract. Many of the cells are sensory and are parts of new neurones taking and carrying the sensory impulses brought in by sensory peripheral nerve fibres.

At the head of the posterior gray horn is seen, under the microscope, a different appearance from the rest of the gray. It is called the gelatinous substance of Rolando, and contains many small cells and a very fine " felt work" of fibres, some of which appear to get into the posterior and lateral columns.

In the region anterior to the gelatinous substance and near the cells of Clarke's column are found most plentifully the so-called Golgi type of cell. (Figure 17.) It is noted for the branching of its processes

Fig. 17.

Fig. 17.—A Golgi's cell from the posterior horn of the spinal cord of a human fœtus, 35 centimetres in length. The star is placed near the axis cylinder or neuraxon. From Lenhossek. Highly magnified.

immediately on their leaving the cell's body, and often showing a remarkable prickly or varicose appearance. The neuraxon soon after leaving the cell body divides dichotomously at right angles, and after suffering one or more further subdivisions terminates in very fine free ends. Lenhossek says that these cells are not found in the spinal cord of reptiles, amphibia and fishes; they are found in the larger mammalians and are most perfect in man.
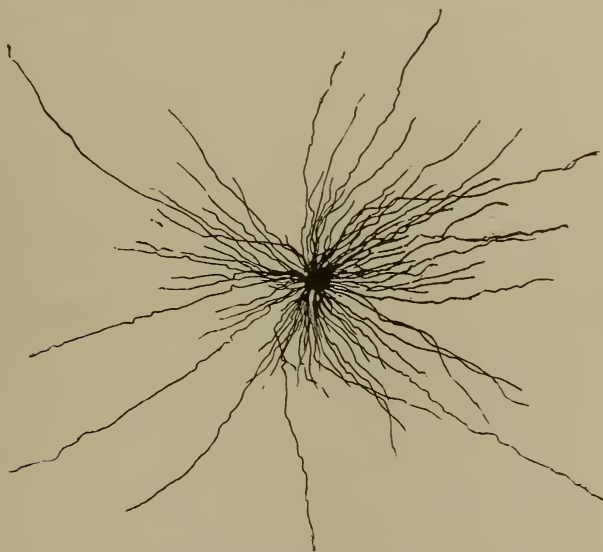

Fig. 18
Fig. 18.—A glia cell from the spinal cord of a child aged 9 months. Lenhossek. Highly magnified.

The supporting tissue of the nervous structures of cord and brain is made up of cells which are small in body with a relatively large nucleus and with many fine processes or outrunners. These exist in great numbers in all parts of the central nervous system, and the interlacing or felting of the processes forms a delicate yet tough support for the nervous elements. They are known as glia cells, or "spider" cells, or Deiter's cells. They are not transformed connective

tissue cells, since they are derived from the ectoderm, and they form the septa, even the posterior central septum being made up of them. According to Len-hossek there is no connective tissue within the central nervous system, except such as may accompany the bloodvessels as adventitia.

The blood supply of the cord may be considered as coming from three great plexuses; the first being the so-called anterior spinal artery, derived as two branches from the vertebrals and soon uniting to a common trunk, continuing downward in the anterior sulcus and receiving reinforcements from the ascending cervical, intercostal, lumbar and lumbo-sacral arteries. The other two are derived above from the vertebrals and are similarly reinforced from the posterior branches of the arteries named. These two plexuses course one on each postero-lateral aspect of the cord. The branches from the three plexuses supply the interior of the cord, those from the anterior artery being distributed chiefly to the gray matter, those from a postero-lateral plexus being considerably smaller and supplying the white matter and part of posterior horn of its own side. All of the arteries of the cord are end-arteries.

The veins of the cord at their beginnings bear no relation to the arteries either in size, distribution or number. The greater part of the blood supply goes through the anterior artery, but the veins for the return flow are larger than the anterior arteries and are situated posteriorly.

The Reflexes.

The reflexes, whose centres are in the spinal cord, are the visceral and peripheral; of the latter there are two kinds, the superficial or skin reflexes, and the deep or tendon reflexes. In all cases a muscular contraction is set up by the occurrence of the related sensory stimulus. It follows that for the production of a reflex the reflex loop or arc, consisting of a sensory nerve fibre, the connections of the same either directly or indirectly with the motor ganglion cell in the cord, the nerve fibre from the same and the muscle itself, must be intact and in physiological order.

Of the deep reflexes, the most important is the patella tendon reflex or "knee-jerk." The mode of obtaining it is as follows: The subject being seated and with one knee crossed upon the other, the patella tendon is struck sharply with the ulnar edge of the examiner's hand, or with a percussion hammer. The sensory impulse thereby aroused is sent to the cord, is passed forward, and arouses in the related ganglion cells of the anterior gray horn a motor impulse which being then sent out produces contraction of the quadriceps extensor muscle, causing the leg and foot to "kick." This reflex is believed to exist in all individuals in a state of health, excepting very young infants, and in some cases in old age. It is absent when any interruption of conducting power

exists in any division of the reflex loop, and when the muscle is atrophic, and thus unable to respond. It is prevented by abnormal conditions of the knee joint (anchylosis, etc.).
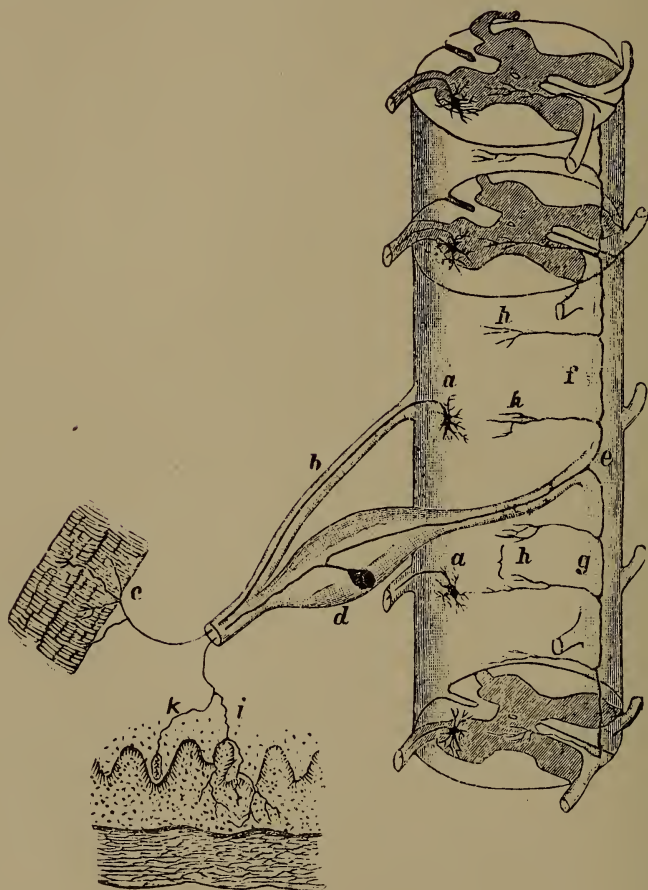

Fig. 19.

Fig. 19.—Schematic representation of the origin, course and ending of a motor and a sensory fibre, as also the relation of the sensory collaterals to the originating cells of the anterior nerve roots. The cord appears transparent. From the motor cell in the anterior horn *a*, arises the anterior root fibre *b*, whose ending as a small brush work on a striated muscle fibre is shown at *c*. In the posterior spinal ganglion *d* (disproportionately enlarged) is seen a single unipolar cell, whose centrally directed process enters the cord as a posterior nerve root, divides at *e* into (*f*, an ascending, and *g*, a descending) main fibres, which above and below turn into the gray and end free, giving off on the way collaterals at *h*. The peripheral process of the posterior spinal ganglion cells passes as the sensory fibre to the periphery, where it ends in a naked arborization as at *i*, or in a complicated structure in a Meissner's corpuscle at *k*. From Lenhossek.

The mechanism in the cord is, however, not inde-
pendent. It is held in check by what is termed in-
hibitory influence from the cortex passing downward
in the pyramidal tracts continually. Hence, when
from degeneration in the lateral tracts, or from brain
disease, the inhibitory influence is obstructed in its
downward passage or is absent, the knee jerk is
characterized by excess in both the amplitude and
rapidity of its motion; it is then said to be exaggerated.
Conditions causing exaggeration may exist with those
for its failure, and then the knee jerk does not occur.

Since in disease of the cerebellum and also in total
transverse lesions in the upper dorsal region of the
cord or higher, the knee jerk has been repeatedly ob-
served to be lost, it has been argued that the vivifying
influence, so to say, passes from the cerebellum to
the centre for the knee jerk. This view has not
received full acceptance by neurologists.

Next to the knee jerk in importance, is the Achil-
les tendon reflex. It is obtained by slightly pressing
the anterior part of the foot in dorsal flexion and
then with a percussion hammer or similar object tap-
ping the Achilles tendon. Contraction of the calf
muscles follows and extension of the foot upon the
leg occurs. This reflex is believed to exist in most
individuals, but its absence cannot be construed as
necessarily meaning the existence of disease; its ex-
aggeration, however, is of the highest importance.
When degeneration of the lateral columns has con-
siderably advanced, the examiner finds that upon dors-

ally flexing the patient's foot somewhat sharply, the
calf muscles at once contract and then yield to the
continued extension only to immediately contract again,
and so on as long as the pressure is kept up or until
the muscles become fatigued. The result is a strong
planto-dorsal oscillation of the foot at the ankle joint;
the phenomenon is termed "ankle clonus."

Other deep reflexes are those of the elbow and
wrist. When the back of the radius or the back of
the ulna is tapped near the wrist, the result is in
many cases a reflex movement of the forearm laterally
in the direction of the bone so tapped. Similarly,
if the tendon of the triceps at the back of the elbow
is tapped the partly flexed arm becomes extended.
The absence of the arm reflexes is of no importance,
since they cannot be elicited in all persons; their ex-
aggeration has the same meaning as has that of the
patella and Achilles tendon reflexes.

Of the skin reflexes the most important is the
cremaster reflex. It is obtained by stroking some-
what sharply (using the back of the finger end, or
the dull end of a paper cutter, etc.,) the inner aspect
of the thigh. Then follows contraction of the cre-
master of the same side, plainly drawing upward the
testicle. The reaction is very lively in the young,
but it may be normally absent in the old.

The inhibitory influence is increased by the patient's
attention being directed to the procedure. Hence,
when the knee jerk does not appear or is very weak,
by directing the patient to hook together the fingers

of the two hands and to pull hard without letting
them part, the knee jerk will be produced if it had
been previously over-inhibited.

There is a false ankle clonus observed in hysterical
conditions. It differs from the real one by the evi-
dent existence of voluntary or intentional quality instead
of the automatic machine-like movement characteristic
of the real clonus.

Other superficial reflexes are: the abdominal reflex,
in which, when the abdomen is stroked along the
outer edge of the rectus muscle, the muscle contracts;
the gluteal reflex, causing distinct movement in the
gluteal mass when the skin of one buttock is stroked;
the pectoral or breast reflex, and the scapular reflex,
whose names indicate their location.

The plantar reflex is of some importance, but it
has not been sufficiently studied. When the sole of
the foot is tickled lightly, the foot is dorsally flexed;
when the stimulation is strong, the leg is flexed upon
the thigh and often the thigh upon the abdomen.
Absence of this reflex, with exaggeration of the knee
jerk, is taken by Dr. Buzzard as strong presumptive
evidence of a functional disorder.

The illustration, Fig. 20 (from Gowers), shows the
topographical relation between the spines of the ver-
tebræ, the different emerging spinal nerve roots, and
the location within the spinal canal of each theoretical
segment of the cord with its nerve roots, and the
table (from Starr and applied thus by Goldscheider)
gives the location of the different centres, reflex, vis-

ceral, etc., in the cord, as well as the relation of different levels of the cord to peripheral sensory and motor functions.

Fig. 20.

Diseases of the Spinal Cord.

The diagnosis of disease of the spinal cord is based upon a knowledge of its anatomical structure and of the functions of its constituent parts. Variations from the normal in the latter, involve the assumption of changes in the former, changes that may be too fine for discovery by our present methods of research or gross enough to be thus observed. For these two types of disease the terms functional and organic are employed, as in affections of other parts of the nervous system.

An affection of the cord may be limited to a small part of it or may be diffusely spread through a large part; hence the terms focal and diffuse.

Disease may limit itself to certain definite structures of the cord, such as the posterior columns or the cells of the anterior gray horns; such manifestations are termed system diseases.

Disease of the cord may occur secondarily to disease in some other portion of the nervous system, the disease-process simply progressing in one direction or the other until it finally reaches and enters the cord. In this manner locomotor ataxia or degeneration of the posterior columns has been explained as simply the continuation of degeneration of the posterior spinal nerve roots. Injury to the vertebræ, growths within the bony spinal canal, or hæmorrhages acting by pres-

sure, may destroy parts of the cord and give rise to extensive secondary degeneration, both upwards and downwards.

The microbes (or their toxines) may be carried to the cord by the blood-vessels and give rise to inflammatory-degenerative changes. So with many inorganic and organic poisons, when carried by the blood-vessels they exercise a specially pernicious effect on the cord (so-called selective action of poisons).

Disease of the blood-vessels themselves (as in arteriosclerosis, chronic alcoholism, syphilitic end-arteritis and peri-arteritis) by decrease of blood supply and by local irritation from them, can cause inflammatory-degenerative changes in the cord.

Diagnosis of spinal cord disease is first by exclusion of disease of peripheral nerves on the one hand and of the brain on the other; secondly, determining the segments of the cord involved; thirdly, determining the extent in cross section of the cord structures implicated, and finally determining the character of the lesion or disease process.

The nerve fibres forming the white matter of the cord have each an axis cylinder, which in turn is clothed with a sheath of medullary substance, but there is no connective tissue sheath. Herein is the difference between a peripheral nerve fibre and a spinal cord fibre. Upon the existence of the connective tissue sheath and its nuclei depend the possibility of and tendency to regeneration of the peripheral nerve fibre after destructive change has occurred. The

spinal cord fibre destroyed at any part, not having a connective tissue sheath, cannot be regenerated, and the portion of the fibre not in connection with its originating cell undergoes secondary degeneration, which is permanent. The direction in which degeneration advances is that of the impulses normally carried by the fibre. Ascending degeneration, therefore, is found only in the posterior columns, the direct cerebellar tract and the antero-lateral tract; descending degeneration is found in the part of the anterior column occupied by the direct pyramid tract and in that of the lateral column occupied by the crossed pyramid tract.
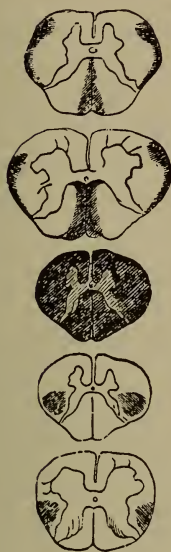

Fig. 21.

From Edinger. A transverse lesion is shown in the middle section; below are shown two sections with descending degeneration in the lateral columns; above are two sections with ascending degeneration limited to the posterior columns and to the ascending tracts at the lateral edge of the cord (direct cerebellar and antero-lateral tracts).

The evidences of degeneration, as shown in properly stained and hardened sections of the cord at successive levels, lessen as the section is further away from the site of the original lesion, owing to the turning inward into the gray of fibres at different levels.

There is also a retrograde degeneration that is towards the originating cell; it is very slow in its progress. As the fibre is already out of function it has no clinical bearing.

Spinal Hæmorrhage. May be outside the cord or within its substance. The former is divisible into extrameningeal and intrameningeal, according to the

position of the effusion, and most often results from trauma (in the new-born from injury during extraction), during convulsive attacks, poisoning by strychnia, during infectious disease.

Symptoms resemble those of meningitis; painful stiffness of the spine, radiating pains from the back into limbs, hyperalgesia, paræsthesias, muscular rigidity. Later, paraplegia with anæsthesia. Small hemorrhages may give no symptoms, large ones are characterized by the sudden onset of symptoms with evidences of shock, the patient perhaps falling to the ground without loss of consciousness. The symptoms advance rapidly to their full height and amelioration is slow. Chronic meningitis may be a sequel.

Hæmatomyelia or hæmorrhage within the substance of the cord may occur primarily from disease of the blood vessels or from tumors, myelitis, etc., or, secondarily, from trauma, excessive muscular exertion, or convulsions. The symptoms are paralysis of sudden onset, usually paraplegia, but if the lesion is high up the arms are paralyzed. Sensory disturbances, except some anæsthesia, are slight, if at all, but paralysis of the bladder and rectum are to be expected. Patellar reflex may be lost at first, to return later and become exaggerated, except when the lumbar cord is the seat of the hæmorrhage, in which case it is permanently absent.

The hæmorrhage may set up a myelitis (or may be the result of it).

The prognosis in both forms is serious in cases at all severe; in hæmatomyelia the chances of restored integrity to the cord are but few.

Spinal Meningitis.

Within the spinal canal, yet outside the proper structures of the cord, lesions of different kinds can occur.

Hæmorrhage may be extrameningeal or intrameningeal; the former is rare and occurs chiefly through trauma; the latter through trauma or in the course of spinal leptomeningitis. The symptoms in either case are those of meningitis, the diagnosis, when not evident from traumatic influence, being made from the suddenness of onset of the symptoms.

Meningitis of the cord, like the meningitis of the brain, is divisible into pachymeningitis and leptomeningitis, according as the dura mater or the pia mater is the membrane chiefly or solely affected.

Since the roots of the spinal nerves pass through the meninges in their course to or from the cord, it is evident that inflammation of the membranes must inevitably affect by irritation and pressure those structures; hence all forms of meningitis are characterized by certain symptoms in common, viz., eccentric or radiating pains from the spine, hypertonicity of muscles, especially those connected with the vertebræ, hyperalgesia, paræsthesias and muscular twitchings in the limbs. Increasing exudation or thickening of the membrane may be great enough to cause paralysis with atrophy in one or more limbs.

Pachymeningitis spinalis externa does not occur

primarily, but only from external injury or more commonly from extension of disease of the vertebræ, etc.
The symptoms already mentioned prove the involvement of the membrane, while the site of the primary disease determines the course of the new symptoms.

Pachymeningitis cervicalis hypertrophica. A special form of inflammation of the dura, of slow course, occurring in adult life and chiefly in males, in the cervical region of the cord and usually in its lower portion. The causes assigned are alcoholism, injury, exposure to cold, and syphilis.

Beginning as a pachymeningitis interna hæmorrhagica, the effused blood organizes, new hæmorrhages occur from the weak vessels of the new tissue, and this process being repeated from time to time, a succession of layers unites the dura and pia together, forming a dense structure constricting nerve roots and compressing the cord structures.

The disease is divisible into three stages. First: the irritation of nerve roots causes pain and stiffness of the neck, with violent pain radiating to the occiput and down the spine and the arms, aggravated by motion and pressure; in some instances the pains (and paræsthesias) are limited to one side or nearly so. Second: after several months the stage of paralysis with atrophy begins. When the affection is in the lower part of the cervical cord, its manifestations are in the ulnar and median distribution in the arms. The unaffected extensors retract the hand, the fingers being flexed. If the meningeal thickening does not extend below

the origin of the seventh cervical nerve, the ulnar and median distributions escape in great part, while the radial distribution being more greatly affected, the long extensors are paralyzed and there is a condition similar to wrist-drop. (Ross.) Third: the stage of spastic phenomena begins when the compression of the cord above has become great enough to affect the lateral columns. Hence the usual symptoms of spastic paraplegia.

The disease may last for years, at times with periods of remission. The prognosis is always serious, but a few cures have been reported. The diagnosis, where the site of the disease is in the cervical enlargement, is not difficult, since the distribution and character of the pains will exclude progressive muscular atrophy, amyotrophic lateral sclerosis and myelitis. The history of the case will exclude Pott's disease. When the symptoms are predominently uni-lateral it may be difficult to exclude tumor, but in such case the lesion is to all intents a tumor.

Leptomeningitis spinalis. Inflammation of the pia mater, as an acute disease, exists only secondarily to some other affection, as by extension of similar trouble in the membranes of the brain or by transmission of infectious material as in pyæmia, tuberculosis, the infectious diseases, of which the epidemic form of cerebro-spinal meningitis is one; in the latter, instances have been reported in which only the spinal membrane was apparently involved.

The disease may begin after some exposure, with

a chill followed by fever, pains in the back and radi-
ating pains in the limbs, with painful stiffness of the
back and neck, rigidity of the muscles of the back
or even opisthotonus. The pains are aggravated by
even passive movements of the limbs, and besides ten-
derness of the skin to touch, any pressure on the
deeper parts is provocative of great pain. The tendon
reflexes are exaggerated in the early stage. The
muscles of the abdomen may be hard and contracted
from spasm, and in some cases tightness of the chest
amounts to dyspnœa. Similarly, from spasm, the urine
may be retained and obstinate constipation exist.

The disease progressing, the amount of exudation
acts more by pressure, and paralytic symptoms ensue,
commonly paraplegia with anæsthesia and paralytic
symptoms on the part of bladder and rectum.

The disease may last a few days or some weeks;
death may be preceded by a considerable rise in tem-
perature and may occur from the effects of bedsores
(sepsis) or from those of cystitis (pyelo-nephritis) or
from exhaustion.

The exudation is commonly purulent or sero-puru-
lent, the membranes being bathed in it. If absorption
take place, the membranes may be united to one
another and to the cord, while increase of connective
tissue with subsequent contraction may give rise to
secondary changes in the nerve roots and the cord,
thus producing a chronic meningo-myelitis, or menin-
gitis alone may follow.

The diagnosis is to be made from that of myelitis,

rheumatism, tetanus and meningeal hæmorrhage. In a pure myelitis there is little or no pain and the paralytic symptoms predominate; in rheumatism there are no radiating pains and no spasm of distant muscles; in tetanus the early appearance and prominence of trismus, as well as the absence of increased temperature, are decisive; with meningeal hæmorrhage there are radiating pains, but they appear with great suddenness and violence.

The prognosis in internal spinal meningitis is always grave; the higher the fever, the earlier paralysis sets in; and the more severe the pains, the worse is the outlook. So in tubercular cases the prognosis is worse than in rheumatic ones and better in traumatic cases than in those arising without known cause.

Treatment. Absolute rest in bed must be enjoined with freedom from external irritation of all kinds, even including light and noise. In traumatic cases the well-known remedies, *Arnica, Hypericum perforatum, Rhus toxicodendron;* in cases following exposure to cold or wet, *Rhus toxicodendron, Dulcamara, Bellis perennis,* will be thought of, the selection to be made according to the special symptoms in any one case. *Belladonna, Bryonia alba, Cicuta virosa, Nux vomica, Mercurius, Physostigma venenosa* and *Iodoformum* should be studied.

Iodoform has repeatedly caused cerebral meningitis when absorbed from wounds, and its use in the 6th dilution by the writer has more than once been remarkably helpful in that form; hence it is advised in the spinal form.

Chronic Spinal Meningitis. A chronic idiopathic leptomeningitis spinalis is an extreme rarity, if it exist at all.

As the final stage of acute spinal meningitis, or as secondary to syphilis or alcoholism or injury it is not unknown. Its symptoms are those of the acute form, but in less severity, and the anatomico-pathological changes in the membranes are increased connective tissue proliferation, causing adhesion of pia and arachnoid, with thickening and opacity. There may be adhesion to the cord causing compression and inflammation of its circumferential edge.

When the affection is the result of the syphilitic processes, the more characteristic signs are stages of advance and regression of the symptoms; the in-completeness of the phenomena and the extensive-ness of the manifestations would give strong pre-sumptive evidence of such cause even without satis-factory history.

Treatment. If the case is believed to be syph-ilitic, *Syphilinum*, high, may be given, or *Mercurius*, *Aurum muriaticum natronatum* or *Nitric acid*, all low, may be used. In non-syphilitic cases the great an-tipsorics, *Sulphur*, *Calcarea carbonica*, *Lycopodium clav-atum* and *Silicea* ought to be held in mind. *Iodo-form* 6 has been of such signal value in meningitis that even in old cases, where it has not been used, its employment should not be omitted.

Myelitis.

The word myelitis means an inflammation of the spinal cord, and, strictly speaking, might be applied to any inflammation of any part of that structure. Its use, however, without any qualifying word, is commonly held to apply to inflammatory processes extending diffusely through the cord transversely. It may include the whole cross-area of the cord, or only a part; it may extend longitudinally through many segments of the cord, or include only one. More than one focus of inflammation may exist at the same time.

The anatomical changes resulting from the myelitic process are softened consistency of the cord, loss of difference in tint between the white and gray matter, while under the microscope are seen increased vascularity, the vessels being surrounded by leucocytes, swelling of the axis cylinders and of the myeline, and swollen and deformed nerve cells. In a late stage there are evidences of degeneration of the myeline, aggregations of granular corpuscles and of fat granules, and still later, proliferation of the neuroglia, leading to a final stage of connective tissue formation, with disappearance in great part of all nervous elements.

At present the process is held to be dependent upon infection, at least in the majority of cases, but exposure to cold and wet, traumatism, etc.,

cannot be denied as occasional causes. The infectious diseases, syphilis, gonorrhœa and septic processes are the chief excitants.

The symptoms of myelitis are those of interference with, or loss of function in, the different structures of the cord and vary with the varying height and extension of the inflammatory process, and its varying intensity in the cross section of the cord.

The typical form is a transverse myelitis, and the most common site is the dorsal cord.

Symptoms. A prodromal period of some sensory disturbance, limited to slight paræsthesia or pain in the related limbs, is followed by weakness or heaviness in the legs that soon becomes paralysis, and anæsthesia for all kinds of sensation, the sensory loss extending upon the trunk as high as the nerve distribution from the upper limit of the disease in the cord. Just above this limit there is irritation and consequent hyperæsthesia upon the trunk, and at its junction with the anæsthesia is felt the very common girdle sensation.

With the paraplegia are associated stiffness of the legs and increase of the tendon reflexes, while later, spasms or more prolonged flexor contractions in the lower limbs may occur. If the disease is intense in quality and involves the whole cross section of the cord in the upper dorsal region or higher, the tendon reflexes are abolished in many cases (Bastian, Bruns).

Paralysis of bladder and rectum are partly due to anæsthesia, partly to interference with the voluntary

control. In the early stage the bladder requires the use of the catheter; later there is incontinence for both viscera.

Trophic changes may be found even early, due to the influence of uncleanliness and the lessened resisting power of the skin; bed-sores result at points most under pressure, such as the sacrum, trochanters or heels, and carry with them the danger of septic infection.

Cervical myelitis. The symptoms already described are present, with the difference that the higher site of the trouble in the cord causes a higher limit (on the periphery) to the anæsthesia and the girdle sensation, and gives rise to paralysis in the arms, usually with some amount of degenerative atrophy. Dyspnœic conditions may be present from implication of the respiratory muscles.

Lumbar myelitis. When the lumbar enlargement of the cord is the site of the lesion the symptoms are different. The paralysis of the lower limbs is degenerative, the tendon reflexes are weakened or lost, anæsthesia may not extend higher than the inguinal regions, and paralysis of bladder and rectum are very pronounced. The skin reflexes are lessened or absent.

In myelitis of any region of the cord vaso-motor changes may occur; cyanosis and œdema of the paralyzed limbs are not infrequent.

The course of an acute myelitis is rather rapid, so that in a few days, or perhaps a week, the

symptoms have fully developed, with some increase of temperature; then follows a stationary period lasting some weeks or some months, and from this the disease may regress to cure or to improvement only, or advance to a fatal ending, or to a stage of chronic myelitis, with more or less permanent retention of many of the foregoing symptoms.

The prognosis is always doubtful, but is more favorable when the disease follows one of the acute infectious diseases; when occurring during the puerperal state, or during tuberculosis, or from sepsis the prognosis is bad. The presence of radiating pains is considered by Oppenheim of good augury, in doubtful cases, while the early appearance of bed sores, or of complete paralysis of bladder and rectum are evil omens.

Chronic myelitis, except as the outcome of the acute form of the disease, is uncommon. The diagnosis must be made by the symptoms already described appearing slowly and with less intensity.

Syphilis of the Spinal Cord. That syphilis is often a prominent factor in the etiology of myelitis cannot at present be doubted, but as the disease process only affects the cord by way of the pia and arachnoid, the result is more properly a meningo-myelitis syphilitica. The anatomical changes seen in the meninges at the autopsy may, indeed, be slight, while the cord structures are severely affected and present the appearances of a diffuse or disseminated myelitis; in other cases the cord is involved only at its periphery (in cross section).

The blood vessels are both starting points and channels for extending the disease process, and not only endarteritis, but even obliterating phlebitis may be present.

The manifestation of the disease may occur within a year after infection, oftener within the first six years.

The symptoms vary, as in all diseases of the cord or meninges, according to the height of the diseased segments, and the extent of the process in cross section. Symptoms of irritation and compression of the nerve roots are radiating neuralgic pains in back and extremities, girdle pain, anæsthesia and paræsthesia and, if the cervical or lumbar enlargement is specially affected, atrophic paralysis in arms or legs, limited to single muscles or groups of muscles, may be expected. From the implication of the cord structures paraplegia occurs, often with predominance of the paretic state in one limb, or a Brown-Séquard's paralysis, with loss of control of the sphincters.

More characteristic of the syphilitic process is the variability of the symptoms from time to time, or at times from day to day, even the tendon reflexes partaking of this.

In some cases there is a marked spastic gait, with exaggeration of the knee jerks, yet without great increase of muscular tonus. As sensation is in these cases (called by Erb syphilitic spinal paralysis) but slightly impaired, it may be assumed that the brunt of the attack is borne by the cord itself.

Compression Myelitis. Compression of the cord can occur as a result of meningeal thickening (q. v.), hæmorrhage within the spinal canal (q. v.) of tumors, in the ordinary sense, growing within the vertebral canal and of deformity of the spinal column, most often as a result of tuberculosis or caries of some of the vertebræ (Pott's disease).

In the process the bodies of some of the vertebræ are changed, softened and disappear, with consequent falling together of the less affected vertebræ above and below, causing angular projection of the spinous processes of the diseased vertebræ. The included cord is of course bent and stretched and also exposed to pressure. Additional pressure will follow if the disease involve the periosteum, or cheesy pus accumulate beneath the latter (sometimes for several inches). In other cases formation of pus may be extensive upon the dura mater, causing additional compression.

The symptoms are of two kinds: those of the vertebral disease and those of the spinal cord and nerve roots. The former are stiffness of the spine, pain on movement, tenderness of certain vertebræ to pressure (a symptom to be accepted with great caution, as it is found in neurasthenia) and protrusion of some of the spinous processes. Even the latter is not to be taken as evidence of vertebral disease, for Charcot has shown (Iconographie de la Salpétrière, Vol. I.) photographs of the ancient Greek statues of athletes having marked projection of the vertebral spines in

the lumbo-dorsal region. The writer holds this to
be due to slight displacement backward of the pro-
jecting vertebræ, and has seen it after severe work
and in some cases of neurasthenia.

An angular protrusion is different, and may be ac-
cepted as diagnostic of disease of the bodies of the
affected vertebræ.

The vertebral symptoms may exist for a long time
without attracting much attention.

The first spinal symptoms are usually painful sen-
sations radiating along the course of certain spinal
nerves, whose roots are in the extent of the lesion,
and they vary according to its site. If the cervical
vertebræ be affected the pains are in the shoulders
and arms; if in the dorsal vertebræ there is a girdle
pain. The pains are mostly neuralgic in character,
but at times are dull, drawing.

The tendon reflexes whose reflex loops are below
the site of the disease process are always increased.

Next, weakness and stiffness of the lower limbs
appear, the weakness gradually increasing until com-
plete paralysis results. These symptoms may begin
in one leg, to appear later in the other, or may pre-
dominate in one. The seat of the affection is in the
majority of cases in the dorsal vertebræ; when in the
cervical vertebræ the arms are affected first and the
lower limbs only later in the course of the disease.

In many cases the spastic paraplegia, with rigidity
of legs, ankle clonus, etc., are present, due to the
interference by compression with the passage of in-

hibitory impulses down the cord. In some cases the paralysis of the lower limbs is a lax one.

Sensory disturbances, besides the pains already mentioned, are limited to some anæsthesia and paresthesia. Bladder and rectum are affected in nearly all severe cases of compression of the cord, in the early stages retention of urine appearing, in the later stages incontinence.

In the few cases in which the lumbar vertebræ are the seat of the disease there will be atrophy of the related muscles with reaction of degeneration.

The disease is almost always chronic in its course, and the vertebral affection may exist for months and even for years before the spinal symptoms appear. Sometimes the spinal symptoms give the first clue to the existence of disease of the vertebræ; often both sets of symptoms appear about the same time and in some cases the deformity of the spinal column is absent.

The further course of the disease is protracted, often with periods of apparent cessation or even of temporary relief of the paralysis.

The majority of cases die, bedsores, cystitis and pyelonephritis, septicæmia or general tuberculosis being agents in the fatal ending. The prognosis is much more unfavorable when the bone affection includes either the lumbar or cervical enlargement of the cord, or when it appears in the adult, or when evidences of general tuberculosis are present.

Diagnosis. First, from the symptoms already given,

diagnosis of pressure upon the cord in some definite portion must be made. Next in order is the diagnosis of the character of the latter. Pott's disease appears in young life, although it may come at any age. Carcinoma of the vertebræ appears for the most part after the 40th year. In the one case there often are signs of tuberculosis, in the other the existence of carcinoma elsewhere would be a guiding point. In Pott's disease the kyphosis is as a rule angular; in carcinoma the deformity is a protuberance not angular and less in size than in the other condition. In carcinoma of the vertebrae the initial sensory symptoms are of great intensity, so that the term paraplegia dolorosa is used to designate the extremely painful condition found in compression of the cord, due to this cause.

Pathological anatomy. Post mortem, the cord when taken out is noticed to be softer than normal in the compressed portion, is smaller and sometimes even appears constricted. There is no real inflammation, so that the term myelitis is incorrectly applied to the condition present. Microscopically, a number of the nerve fibres are destroyed, many are in the process of degeneration, with swollen axis cylinders already deprived of their myeline sheath. Later comes proliferation of glia tissue, and subsequently a sclerosed area.

The diagnosis of myelitis will depend upon the existence of paraplegia, stiffness of the limbs, exaggeration of the tendon reflexes (occasional loss of knee jerks in a total transverse myelitis high up in

the cord), anæsthesia in the paralyzed parts, girdle sensation at the upper limit of anæsthesia, paralysis or paresis of bladder and rectum, the occurrence of bed-sores. Meningitis is characterized by pain and superficial tenderness of limbs without bladder symptoms; multiple neuritis, by pain along the nerve trunks and no bladder trouble. Landry's paralysis of central type is a rapidly ascending disease of the cord.

The hygienic treatment of myelitis consists of measures to prevent, if possible, the formation of bed-sores, or the occurrence of cystitis. For the first the patient should be often examined in the parts exposed to pressure, and these should be bathed in alcohol and water, while the bed clothing should be scrupulously clean. If there is paralytic retention of urine, the catheter must be used only after extreme aseptic precautions; if there is incontinence, a suitable urinal should be kept always in place. The bowels should be relieved by the aid of plain enemata.

Of the remedies to be prescribed, those having causal indications should be used first. For traumatic cases, *Arnica*, *Bellis perennis* and *Rhus toxicodendron;* for cases following exposure to dampness or wet cold, *Dulcamara*, *Rhus toxicodendron*, *Bellis perennis.* A cause should always be looked for. When none of the foregoing is found, the existence of some infectious disease (or a wound) may give a clue to an effective remedy. If syphilis has existed, and no other cause is assignable, *Mercurius iodatus flavus*, or *solubilis*, may be prescribed, or the isopathic prin-

ciple may be employed by prescribing *Syphilinum* 200th or higher. Similarly, if gonorrhœa has preceded the disease. Many substances, metallic, vegetable or animal in origin, have caused experimentally, myelitis (Bruns) in the lower animals, but our provings have not been carried to such extent on the human subject. Nevertheless many of our drugs have among their symptoms those strongly characteristic of the disease. The symptoms that are diagnostic are not, as we learn from Hahnemann, the ones leading to the selection of a remedy, but rather those that are striking and peculiar, together with the aggravations and ameliorations.

In the early stage, *Aconite, Belladonna, Gelsemium, Veratrum viride*; when any fever has lessened, *Arsenicum*, the metals, especially *Mercurius, Cadmium* and *Plumbum. Oxalic acid, Secale* and *Phosphorus* may be studied.

In the chronic condition, besides the remedies mentioned, *Berberis vulgaris* when the bladder symptoms are marked, *Æsculus hippocastanum* when rectal, sacral or perineal symptoms are prominent, *Picricum acidum* when the genito-spinal centre is irritated. In old cases, with marked spastic symptoms, *Lathyrus sativus* has been of great service.

Cases of myelitis uncomplicated with meningeal involvement are not common, and the reader is referred for additional remedies to the section on spinal meningitis.

System Diseases of the Spinal Cord.

Certain systems of fibres, or of cells, may be attacked by certain degenerative disease processes to the exclusion, or nearly so, of all other parts of the cord. The term does not include processes of degeneration following and resulting from a lesion in some part of a system, such, for instance, as the secondary degeneration of a lateral column after hæmorrhage in the cerebral motor tract.

Poliomyelitis anterior acuta. This disease is predominantly one of early childhood, most of the cases appearing during the first, second and third years, while after the fifth year cases are few, although the disease may occur in the adult.

It is an infectious disease, appearing sometimes as an epidemic in small areas of country, but its special microbe is as yet unknown. It also occurs after other infectious diseases, such as scarlatina, measles and whooping cough.

Symptoms. The outbreak of the affection is like that of any acute infectious disease. The patient is taken, usually while in good health, with high fever, headache, some pains in the limbs, vomiting at times, and may become delirious or stupid, and in some instances will have general convulsions. These acute symptoms last from a few hours to a day or two, but occasionally may continue a week or so.

At the end of the acute stage it is noticed that the child is paralyzed, the paralysis being widespread and, perhaps, involving all four extremities, the trunk muscles and the bladder. A slowly progressive improvement soon sets in, and whole limbs may be restored to full power and activity, or many groups of muscles regain their function. This process may continue for several weeks, or for some months, and then it stops, and the patient is left with paralysis of certain groups of muscles, most frequently in one leg, very often in both, less often in an arm, or in an arm and leg.

The paralysis is a lax one, with loss of the tendon reflexes of the muscles involved, and with atrophy and reaction of degeneration.

The muscles most commonly left in this state are, in the leg, those on the extensor side, the anterior tibial, the common extensors and the external peroneal; any one of these or any two or all three may be affected. In the thigh, the quadriceps extensor and the inward rotators are often the ones chiefly paralyzed; in the forearm, the extensors; in the arm, the deltoid and flexors. I have seen one case in which the calf muscles were completely atrophied.

Sensibility is at no time altered, nor is any pain present, except some tenderness of muscles during the process of atrophy.

Owing to the unopposed action of unaffected muscles, various deformities arise, talipes equino-varus, talipes valgus, flexor contraction at the knee, and some lateral

curvature of the spine being those most frequently seen.

Pathologically, the disease is a myelitis limited to the anterior gray horns of the cord, with destruction of some of the motor cells. As a cell once destroyed cannot be reproduced, it follows from the regression of the paralysis that many of the cells have not lost their integrity; and cases of paralysis, from the disease under consideration, existing for some years, have shown very great improvement as soon as treatment was begun, which continued until the limb or limbs were practically restored to usefulness.

The nutrition of a limb severely affected is lowered and its rate of growth retarded. Hence, in cases of long standing, the bones are smaller than normal in all directions and the member is shorter, while the skin is cyanotic and cooler than that of its healthy fellow.

The diagnosis in the acute stage cannot be made, except in the presence of an epidemic of the disease. When the paralysis is first observed, if convulsions have preceded it, the physician may think of cerebral infantile paralysis; but in poliomyelitis the loss of the tendon reflexes would resolve all doubt. From multiple neuritis the absence of pain or of tenderness along the nerve trunks, and the fact that multiple neuritis is extremely rare in children, would be sufficient for differentiation.

The chronic form of poliomyelitis anterior is a rarely observed disease. It begins with weakness of

one limb, which gradually increases until the use of the member is seriously impaired. Next, the fellow member is attacked, and still later, the other limbs; and in the course of several months there may be incomplete paralysis of all. The paralysis is a lax one, and upon testing there will be found degenerative atrophy of certain groups of muscles, just as in the acute form. Sensation is not impaired, but there may be slight rheumatic pains. Fibrillary contractions in the degenerating muscles are almost always present. The sphincters are not affected.

The disease process, after having gone on for several months, may stop and a process of restoration set in that at times becomes complete. In other cases there is a steady advance of the trouble until all the muscles of the limbs, as well as some of the trunk, are wasted, and the patient is bed-ridden. Death occurs in from one to three years, usually from implication of the respiratory muscles, when pneumonia (from the invasion of the larynx and bronchi by food) or even a slight bronchitis, is sufficient to cause a lethal ending.

Diagnosis. From progressive muscular atrophy of spinal origin the disease is differentiated by the fact that weakness and paralysis precede the atrophy, and that the progress of the disorder is here rapid.

The treatment of poliomyelitis anterior in the acute stage must be purely systematic, as in the case of any infectious fever of childhood; but when the acute symptoms have passed off and the paralyzed condition of the limbs is recognized, *Plumbum* should

be given in a moderate dilution. This metal has caused degeneration of the cells in the anterior gray horns of the cord, and the earlier the diagnosis the sooner the remedy can be prescribed. After the active process has ceased in the cord, restoration of cells not destroyed takes place to a considerable degree, and it is fair to assume that an early use of so homœopathic a remedy as lead is, will be of great value. This remedy is of undoubted service in cases of old standing paralyses from a previous poliomyelitis. The writer gives it in 30th potency, a dose every night for a week, intermits for a week and resumes—and so on. If other remedies are indicated, they should be given from time to time.

Goldscheider has shown that the trophic influences governing the peripheral parts are kept active by the stimulus of centripetal impulses from the periphery. Hence, the undoubted value of massage, of "salt rubbings," etc., in this paralysis. But greatest of all is the systematic use of galvanism to the affected muscles. The anode is to be used with an interrupting handle, and a current strong enough to cause contraction in the muscle is to be passed and then broken, making interruptions and contractions of the muscles about twice in a second, twenty-five or thirty times. Another muscle or set of muscles is then treated, and so on until all the paralyzed muscles have been thus exercised. The other pole, cathode, is to be kept on some indifferent part, and possibly with advantage over the spine above the

site of the cord lesion. This should be a large flat electrode. The current required to cause contraction may be so great that the pain cannot be borne, in which case by employing a non-interrupting handle the anode may be used labile, up and down the affected muscles for three or four minutes each, the current strength to be moderate, 6 to 8 m-a. The treatment should be given every other day for a period of 5 or 6 weeks, and then intermitted for a week or two, to be resumed for another period of 5 or 6 weeks. Contractures and deformities may, in the young, be prevented or lessened by orthopædic mechanical devices.

Primary Degeneration of the Motor Path. Under this term Möbius makes a bold generalization and includes the diseases known as spinal progressive muscular atrophy, progressive bulbar paralysis, and amyotrophic lateral sclerosis. Charcot had given to the latter form its place as a distinct type of disease, while Gowers in his consideration of chronic muscular atrophy (text book) says he has not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts were not affected, and in another part of the same chapter he states: "Atonic muscular atrophy is, at least in many cases, the visible expression of a tendency to decay of the whole motor path, from the cortex of the brain to the muscles."

Since in both of these forms of disease the symptoms of progressive bulbar paralysis often appear, and since the cranial nerve nuclei in the medulla are con-

sidered as the analogues of the groups of ganglion cells in the anterior gray horns of the cord, the generalization of Möbius need not be considered strained, and it is of value in giving a readier insight into the features of the three forms.

Möbius says that a primary degeneration of the motor tract may exist throughout its whole extent, from its beginning in the cells of the central convolutions of the cortex to its ending at the ganglion cells in the anterior gray horn of the cord; that it may involve only the fibres of the tract in their spinal extent and the anterior horns; and, finally, that it may affect the anterior horns alone. Destruction of the cells here of course involves destruction of the rest of the peripheral neurones and the muscle fibres to which they are distributed. The same author doubts the existence of a primary degeneration limited to the lateral columns, that is to say, without implication of the cells of the anterior horns.

The anatomical changes are always the same: the cells become shriveled in appearance, they break up and disappear, as do the axis cylinders, the medullary sheaths being seen in all stages of degeneration; slight secondary proliferation of the glia, slight changes in the blood-vessels, shrinking of the muscle fibres, with fatty or waxy degeneration and fissuration in them, and increase of connective tissue or of fat amid them, and increase of the muscle nuclei, are later changes.

The cause of the disease is unknown, but Möbius thinks that congenital weakness in the neuro-muscular

apparatus is a fair presumption; it begins in the adult as a rule while in good health and is not found in several members of a family.

Spinal progressive muscular atrophy begins usually in one upper limb, more often the right. In the majority of cases, weakness and wasting are noted first in the muscles of the ball of the thumb, in some instances preceded by slight rheumatic pains or numbness. Soon other muscles become affected and the eminence at the ulnar side of the palm or the interossei and lumbricales gradually disappear. Next, in most cases, the muscles of the forearm are involved, but in some instances they escape for a time, the deltoid and other muscles about the shoulder being attacked instead. Occasionally, the atrophy begins in the shoulder muscles.

After the disease has progressed to some extent, the other side is affected, but not necessarily in the same order.

When the wasting in the interossei and lumbricales is pronounced, and the extensors and flexors of the forearm are still intact, the hand assumes the much pictured "claw hand" type; in other cases the unopposed action of the long extensor of the metacarpal bone of the thumb brings that member up to the level of the other metacarpal bones and gives rise to what is known as the "ape hand."

After the deltoid, the triceps and biceps and supra and infra-spinatus are involved in varying order, followed by the trapezius, rhomboid, pectoral, serratus

and other deep muscles of back. The muscles of back of the neck may be affected so that it is difficult to erect the head upon the spine. The intercostals suffer in some cases; in others the diaphragm—in either case respiration is abnormally performed.

The lower limbs are not affected until late in the disease, and usually not so severely as the upper ones.

During the process of wasting the muscles show certain symptoms, not, however, characteristic of the disease, but rather of atrophic degeneration. Fibrillary contractions or "flickering," to use Gowers' term, in parts of a muscle, are quite common. The electrical reactions vary according to the rapidity of the wasting as well as its degree, so that the electrical contractility may be only lessened or it may be extinguished; where there is some amount of paralysis preceding atrophy, as occurs in some parts (Gowers) there may be the typical reaction of degeneration; in other parts there may be partial reaction of degeneration. The tendon reflexes disappear as soon as the related muscles become even slightly affected. Bladder and rectum are not involved, but sexual power is often lost.

The progress of the disease is usually a steady one, at times with apparent cessation, and Gowers holds that when once actually arrested, it does not usually again become active. Its duration may be a few years or many. Death occurs, commonly, from some intercurrent pulmonary disorder or from the invasion of the medulla by the disease process, which, indeed, is

the most frequent "complication," and will be described under diseases of the brain-axis.

Amyotrophic Lateral Sclerosis. It may be accepted that in the great majority of cases of spinal progressive muscular atrophy, the lateral pyramidal tracts in the cord are also degenerated, and that the usual clinical evidence of the latter process is not present, because in all cases the ganglion cells are so extensively involved that the resulting widespread atrophy of muscle renders spastic symptoms with exaggeration of the tendon reflexes impossible. In some cases, the damage to the ganglion cells not being so extensive, the symptoms due to disease of the lateral columns do appear, and then to the syndrome the term amyotrophic lateral sclerosis is applied.

The symptoms begin in the same way as in the previously described form, by gradual spread of atrophy in the small muscles of one hand, but in the muscles not affected there is hypertonicity with active tendon reflexes, the knee-jerks even at this early period often being exaggerated. After several months the patient complains of weakness in the legs, and soon the typical spastic gait with weakness of legs and marked increase of the knee jerk and often ankle clonus, with absence of pain or sensory disturbance and of bladder or rectal trouble, prove plainly enough the existence of degeneration of the lateral columns. The patient can walk until a late period in the disease, but the weakness and stiffness of the legs make walking an effort. The last stage of the disease is the appearance

of bulbar symptoms. Death occurs from some inter-current affection, mostly of the respiratory organs, or from cardiac or respiratory paralysis, due to the bulbar involvement.

The diagnosis of spinal progressive muscular atrophy is not difficult. By the inexperienced it may be confounded with one of the muscular dystrophies. But the beginning of the trouble usually in the small muscles of one hand, the age of the patient at the time of its first observed symptoms, the absence of similar cases in the same family, the presence of fibrillary contractions and in some muscles of the reaction of degeneration, give the stamp of authenticity to the diagnosis.

Chronic poliomyelitis anterior attacks several limbs, the atrophy is preceded by paralysis, the reaction of degeneration is present in the affected muscles, the progress is relatively rapid up to a certain point, when it ceases and improvement results, or else it advances in a way not to be distinguished from progressive muscular atrophy.

The absence of pain or of other sensory disturbances will distinguish progressive muscular atrophy from syringomyelia, pachymeningitis cervicalis hypertrophica and caries of the lower cervical vertebræ.

In the atrophy following lead poisoning there is no advance in progress after the cause has been removed, while the history and concomitant symptoms will decide before such cessation occurs. Amyotrophic lateral sclerosis is readily recognized by its symptoms already

described, and the differential diagnoses just given will apply here. In both forms the occurrence of bulbar degenerative changes are to be expected, and this may lead to confounding the disease with disseminated sclerosis.

If the views of Mobius that progressive muscular atrophy and amyotrophic lateral sclerosis are but the manifestation of congenital fault in the structures of the motor paths, be correct, the problem of treatment becomes more complicated. Goldscheider's view that trophic influence is aroused and kept active by peripheral sensory impressions is widely accepted, but if the cells themselves (motor neurones) are imperfect, increased peripheral stimulation ought to wear them out the sooner. Nevertheless, the writer has seen the most remarkable results, amounting to arrest of the atrophy, in more than one case under the use of faradism to the affected region and of *Phosphorus* 6 internally, continued for some months; and that method is here recommended in the spinal type of progressive muscular atrophy. Other remedies, such as *Plumbum metallicum, Cuprum metallicum* and *Zincum metallicum* may be better indicated in individual cases, and different observers have used *Arsenicum, Picricum acidum, Argentum nitricum, Physostigma venenosa, Belladonna* and *Nux vomica*. The value of the deep acting antipsoric remedies in constitutional conditions may be tested here.

Locomotor Ataxia.

Tabes Dorsalis.   This disease, of frequent occurrence, is, except in a few atypical cases, the easiest of diagnosis among all the organic affections of the nervous system.

Its appearance depends upon the existence of a previous syphilitic infection, or at least Erb's statistics show that in but five per cent. of his cases could a preceding syphilis be fairly excluded.

As the anatomical changes occurring in the progress of the disorder entirely differ from those of the syphilitic process, it is assumed that the latter leaves behind it a toxine having a specially injurious affinity, under certain exciting causes, for certain nervous structures.

The exciting causes are physical overexertion, exposure to wet and cold, an immediately preceding attack of some acute infectious disease, "fast" living and other weakening influences.

From a series of 600 cases of the disorder in Erb's private practice, Leimbach (Deutsche Zeitsch. f. N'h'kunde, Bd. 7, Hft. 5 u. 6) has selected 400 in which the records were exceptionally complete in details.   The first symptom most frequently observed was the lightning pains which occurred as such in 277 cases in the legs, in 5 in the back, and in 1 in the

arms. In 62 cases the lightning pains did not appear
as the first symptom, and in 11 cases only as the
third, while in 47 cases they did not occur throughout
the whole course of the disease, but 100 of the ob-
served cases were examined in the early stage of the
disorder.

A feeling of weakness in the legs and of too readily
occurring fatigue as noted as the first symptom in
78 cases, as the second symptom in 113, as third in
44, and as fourth in 11. Leimbach considers this,
although appearing in many nervous affections, as of
great diagnostic value, especially as an early or initial
symptom.

Loss of the knee jerk and of the Achilles tendon
reflex is the most constant symptom in tabes, yet in
3.75 per cent. of the 400 cases the reflexes in the
lower limbs were normal, but the percentage of such
exceptions will undoubtedly be lessened as time ad-
vances in the 100 cases in the early stage.

Paræsthesias in legs and feet have a high rank as
the initial symptom; they occurred as such in 74
cases, as the second symptom in 110, and as third
in 55.

The disease is predominantly one of middle life,
being most common from 30 to 50, and this because
the syphilitic infection occurs most frequently between
the 20th and 40th years, the interval elapsing between
infection and the appearance of the disease being
usually something less than ten years, although it has
followed within two years and been delayed 15 or 20.

Its occurrence in childhood is denied by Oppenheim, but it may develop as late as the sixtieth year.*

Men are much more frequently affected than women.

The disease is divisible into three stages: the pre-ataxic, the ataxic, and the paralytic.   The first is manifested most commonly as sensory disturbances, consisting of severe sharp pains, lancinating and light-ning-like, shooting down the lower limbs, or limited to small areas, and often mistaken for rheumatic pains. In some cases these are boring or constricting or burning.   They appear in attacks lasting from a few hours to a day or two, and are often intolerable in severity; the intervals of freedom from pain are in-definite in length.

A symptom of great diagnostic importance and which appears early in the great majority of cases, is loss of the reflex contraction of the pupil to light, while the associated contraction in convergence is re-tained.   This manifestation is known as the Argyll-Robertson pupil.   In testing the response of the pupil under the retinal stimulus by light, it is important that the patient keep his gaze fixed on a distant object, for contraction will occur upon converging for a near object, such as the examiner's face.   In a few cases the associated action in convergence may also be lost.   Inequality of pupils may be present with or

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* Mendel has reported some cases of children with undoubted typical symp-toms of tabes, the first appearing at the ages ten and eleven.   In both in-stances there was undoubted syphilitic heredity and in one a vesicular eruption appearing after birth was treated with mercurial baths.   It is now held that early appearing tabes is due to hereditary syphilis.

without the foregoing Argyll-Robertson character, or the latter may be unilateral only.

An early symptom, when it occurs at all, (106 out of 400 cases) is temporary diplopia due to paresis or paralysis of some of the exterior muscles of the eyeball. It comes suddenly without any exciting cause, lasts an indefinite time and disappears; it may return later and then become permanent. The diplopia is often very annoying to the patient, but it may be so slight as not to be noticed except under tests. Ptosis may be present with such ocular palsies or independently; it may be bilateral.

Optic nerve atrophy occurs in about 10 per cent. of the cases (Leimbach's statistics give 6.75 per cent.). It is an early symptom and, indeed, may precede all others. It has been noted in many cases that the disease seems to spend its force upon the optic nerve, for the early appearance of the atrophy is not followed by further development of the disorder, or this is retarded or postponed for years. The atrophy is the so-called primary atrophy, of bluish-gray or muddy tint when seen in ophthalmoscopic examination. It goes on to blindness.

Disturbance in the functions of the bladder is very common in the disease, being found in more than 80 per cent. of the cases, while as first symptom it occurred in 90 out of 400 cases, as second symptom in 119, and as third in 74. The disturbance may be a leakage if the desire to urinate is not at once yielded to, or it may be inability to pass urine

except by the aid of the diaphragm and abdominal muscles.

A paralytic state of the rectum occurs, but much less often than the trouble with the bladder.

Weakening of sexual power and desire, often to complete extinction, was found in 58 per cent., and as a symptom belongs mostly to the early stage of the disease.

The paræsthesias already mentioned as early symptoms of locomotor ataxia are most commonly felt in the feet or legs, especially in the soles and edges of the feet, less frequently but not rarely in the region of anus, perineum, scrotum and penis.   More frequent than the latter are paræsthesias in hands and arms, while a special paræsthesia in the ulnar distribution has been observed often enough to give it a diagnostic value.   The paræsthesias vary in character. Some patients feel as if a layer of felt were under the soles or as if cushions were there, others feel a numbness or prickling or crawling; sometimes a sensation of cold is complained of, and in one case the patient felt as if the sacral, anal, perineal and genital regions were closely packed in ice.   Burning or "raw" sensation is not infrequently felt.

Hypæsthesia or even anæsthesia (often with analgesia), retardation of pain sensation (pin-prick not felt for sometimes two or three seconds), allocheiria (pin-prick in one part felt in the analogous part of the other limb), often precede the occurrence of ataxia.

The ataxia itself is but one form of anæsthesia.

The so-called muscular sense fibres cannot carry the sensory impulse originating in the end organs in the muscles (or in the joints?). Hence the information to the brain of the position of a limb or any portion of it is insufficient and misleading, so that for the successful voluntary motion of any part the needed information must be obtained through the sense of vision. The patient, with eyes closed or in the dark, cannot co-ordinate his muscular actions, cannot stand with feet together without swaying (Romberg's symptom), cannot walk a straight line, and after the ataxic stage has well advanced cannot do these acts even with the aid of the sense of vision. For a time, however, he can walk by keeping the feet widely separated, so as to get a broad base of support, and by throwing the foot, as it were, forcibly, the heel reaching the ground first, thus making sure of each succeeding step. His greatest difficulties are going up and down stairs, and turning around while standing.

When the ataxia reaches the upper limbs it is first observed in the finer movements of the hands, so that buttoning the clothing, writing, or playing a musical instrument becomes difficult, and the latter at last impossible. Ataxia of the arms is relatively infrequent, Leimbach's statistics showing its occurrence in only 17 cases out of 400, and all but two of the cases were those in which the disease was of long standing when the symptom appeared, or else of very rapid progress.

The tests for ataxia are simple enough, and limited

in number only by the ingenuity of the examiner. To have the patient stand with feet close together, eyes being closed, and observe the swaying of the body in different directions; to have him lie down and with one heel endeavor to touch the knee of the other leg, or to put one heel on top of the toes of the other foot; with the foot to describe a circle in the air; to tell him to simply cross one leg over the other—all these are but a few of many possible tests, and in all even slight amounts of ataxia will be discovered by a mere novice. So with the arms. To direct the patient (eyes being closed) to bring the two forefingers together from a distance; to have him touch with one forefinger successively, and each time from a distance, the tip of the nose, the middle of the forehead, the chin, the lobule of the opposite ear, are well-known methods. If ataxia exist, the movements, instead of being done gracefully, as under normal conditions of co-ordination, are made unsteadily, doubtfully, and usually go wide of the mark. The simple test of crossing the legs when the patient lies with eyes closed will often tell the whole story.

Although ataxia is due to impairment of co-ordination, and is not a manifestation of muscular weakness, yet its increase means an excessive amount of muscular exertion when walking, so that, as it increases, the patient becomes more and more unable to make such exertion, and finally, perhaps after years, he becomes bed-ridden, and is in the so-called

paralytic stage. Meanwhile there are other symptoms present.

The girdle sensation, as if a band or girdle were tightly constricting the waist or lower abdomen, occurred in 31 per cent. of 400 cases and even appeared as the initial symptom in 34 of these; it is not diagnostic of this disease, since it occurs in other forms of spinal cord trouble, nor is it continuous, but when present it is extremely trying to the patient.

The so-called "crises" are of great importance diagnostically, but they do not appear to be frequent. Leimbach reports 17 cases of gastric crises (in 10 of these the crises were the first evidence of the disease), 1 case of rectal crises and 3 of laryngeal crises, out of 400 cases of tabes. The crises are suddenly occurring attacks of disturbance in some organ without any recognizable adequate cause. Thus attacks of gastric pain with or without vomiting, sometimes nausea and vomiting without pain; attacks of spasmodic coughing, simulating whooping cough; and attacks of severe diarrhœa, often choleraic in character, are some of the crises. They may last a few hours or many, or even some days, and usually cease as suddenly as they came. Renal crises, consisting of severe pain in the region of the kidneys, accompanied by apparent suppression of renal activity; and clitoris crises or sexual orgasm in the female, have been noted by authors. Möbius classes with the crises attacks of paræsthesia in the rectum, the sensation being that of a thick stick or

similar body being thrust upward. In a case under the notice of the writer this sensation was an almost continuous one for months.

Trophic disturbances occur during the course of the disease in a very small percentage of the cases, and are confined to the bones and joint structures. The bones become fragile, the ends eroded or crumbling, the cartilage may disappear and spontaneous luxation occur, with deformity, due in great part to effusion and swelling, often of the whole segment of the limb, yet without evidences of inflammation. This latter manifestation is known as Charcot's *arthropathie tabétique* and is probably seen more often in hospital cases than in private practice. It affects the knee-joint most often, but other joints may be attacked.

Trophic changes in the skin, such as eruptions of various kinds, may be looked upon as complications, but an intractable form of ulceration occurs at times on the sole of the foot as a result of slight injury, such as a trifling cut.

Neuritis, of degenerative type, in the peripheral spinal nerves, occurs occasionally in advanced stages of the disease; it may lead to atrophy of muscle, most often seen in the legs.

The diagnosis of locomotor ataxia in any well developed case is comparatively easy, the large number of the chief symptoms permitting variation, yet assuring such positiveness in the grouping of them into sets that error is practically impossible. Thus, any one of the following groups makes the diagnosis absolute:

Lancinating pains in legs, Argyll-Robertson pupil and loss of knee jerk.

Loss of knee jerk, optic nerve atrophy, and paræsthesias of feet.

Paresis of eyeball muscles, weakness of bladder, lancinating pains.

Similar groupings of the foregoing symptoms or of others may be made by the examiner, and if in addition there is found Romberg's symptom or ataxia under tests, the diagnosis is beyond doubt. The crises may direct attention to the possible existence of the disease when they occur in the early stage.

Multiple neuritis, after the acute symptoms have passed away, might be mistaken for tabes, since there are often present distinct ataxia, loss of knee jerks, Romberg's symptom, and in some cases optic nerve atrophy as sequel to a preceding optic neuritis. But the history of acute onset, rapid progress, tenderness of nerve trunks, and presence of degenerative atrophy of muscles in the affected limbs, suffice for differentiation. It must be remembered, however, that peripheral neuritis does occur in a few cases of locomotor ataxia.

Post-diphtheritic paralysis (neuritis) may involve the limbs and cause ataxia, loss of knee jerks, paræsthesias, or paralysis of eyeball muscles, but the history, and especially the paralytic symptoms of palate, pharynx and œsophagus as early sequels of the throat affection will prevent confounding the later manifestations with tabes.

Not a few cases have been observed that began apparently as locomotor ataxia, and finally developed into dementia paralytica ; sometimes the reverse has been the case. In both, the eye symptoms may be common, and loss of the knee jerks is found in a not inconsiderable fraction of cases of general paresis of the insane. In doubtful cases time will be needed for determining the diagnosis.

The anatomical changes found post-mortem in locomotor ataxia are first, because most conspicuous (even to the naked eye in most cases), a gray degeneration of the posterior columns, with the exception of a small field anteriorly adjoining the gray at the posterior commissure, and extending laterally somewhat on each side. Next, Lissauer's zone at the end of the posterior gray horn, and the vesicular column of Clarke, show loss of fibres and cells, while the posterior nerve roots show marked atrophy of their constituent fibres.

Just where the pathological process begins has been a fruitful cause of discussion. It has been maintained that a leptomeningitis exists with consequent constriction of the posterior nerve roots as they pass through the membrane and necessarily with neuritis and atrophy. It has been maintained that degeneration of the nerve roots is the starting point without any necessary meningitis. And finally it is held by many that the initial point is degeneration of the cells of the posterior spinal ganglia with extension of the process upward in the cord and downward in the peripheral

fibres. The process attacks at times the spinal root
of the fifth cranial nerve and the respiratory bundle in
the medulla.   The fibres of the tenth nerve or its
posterior nucleus and the twelfth nerve nucleus are
sometimes attacked.

The Gasserian ganglion, which is analogous to the
posterior spinal ganglia since its cells give rise to the
spinal root (so called) of the fifth nerve, in some cases
showed atrophy of its cells and fibres; the optic nerve
atrophy begins in the ganglion cell layer of the retina
and extends backward. The motor nerves of the eye-
ball, especially their nuclei and rootlets, may be af-
fected; the exact cause of the Argyll-Robertson symp-
tom is, however, not yet definitely established, but it
is presumed that some degenerative change exists in
the central gray beneath the corpora quadrigemina.

The changes in the cord first appear in the upper
lumbar segments, except in those cases in which the
disease affects first the arms, when the cervical seg-
ments of the cord are first attacked.

Prognosis. The outlook for cure of locomotor ataxia
is practically hopeless, but in the first and second
stages there is hope of lessening the pains and other
trials of the patient. Even after the so-called para-
lytic stage has set in there is a possibility of amelior-
ating annoying symptoms.

The disease lasts for many years, unless when blad-
der trouble with its consequences occurs early. Death
results from some intercurrent affection.

The treatment of locomotor ataxia must be in the

largest degree hygienic. Severe physical exertion must be avoided, but very moderate exercise in the open air is to be insisted upon. The food should be nourishing and easily digested. Stimulants and tobacco are to be forbidden, or allowed only in the smallest amounts.

The ataxia and also (in some cases) the paræsthesias and pains are lessened by the use of Frenkel's muscle drill or re-education of the muscles. This method consists in the carrying out of definitely ordered co-ordinated muscular actions in which the patient must use the will in directing the muscles. The exercises are as varied as the directions in which his muscular co-ordination is lost, and are meant to utilize to the highest degree the few remaining sensory nerve fibres. It is not applicable when locomotion or the power of balancing is lost, nor when optic nerve atrophy has far advanced, as the eyes must be used to help correct faulty action. The exercises are in no sense gymnastics, strength as such not being sought.

The use of galvanism alone (or combined with faradism) along the spine and limbs is recommended by some observers as serviceable in relieving many of the annoying symptoms; the current should be rather strong, 15 to 25 or 30 m-a. The writer has used it, but has seen no special results.

Treatment by suspension in Sayre's apparatus, modified so that the suspension is from the elbows (arms being flexed) as well as from the head, has in some cases been helpful, especially in relieving the bladder

symptoms. It should be gradual in beginning and ending, should be given every other day, two or three minutes at a time, during six or seven weeks. It is not admissible if arterio-sclerosis or bulbar symptoms are present, nor in the so-called paralytic stage. A mild form of suspension may be given by having the patient seated, so that the suspension shall use only the weight of the body above the hips at first.

The employment of hydrotherapy is recommended by many; the matter is one to be decided only after knowledge of the patient's susceptibility in this direction.

Believing with Erb and Möbius that tabes is a post- or para-syphilitic disease, the writer has during the past few years used the principle of isopathy in treating it, and has given *Syphilinum*, 200 or higher. Marked improvement has been seen in some cases, but this may have been due to the patient's ceasing to use powerful drugs. Ergot has caused sclerosis of the posterior columns, as shown by post-mortem evidence, but *Secale* in different potencies, has not in the writer's hands influenced the disease in the least, undoubtedly because *Secale* was homœopathic only in a gross way; in addition, the cases of poisoning by Ergot showed no progression in the affection produced.

Æsculus hippocastanum is said to have caused the disease; "whole flocks of sheep, having been fed during the winter on horse chestnuts, have been afflicted with Tabes Dorsalis, and many have died of it." *

* Ad. Lippe, M. D., Homœopathic Quarterly, April, 1870. The constriction in the rectum, which has been observed by the provers of *Æsculus,* is often present in this disease.

Four cases of cure are recorded by Bönninghausen under the use of *Aluminum metallicum*. Other observers have used with asserted benefit *Argentum nitricum, Phosphorus, Plumbum metallicum* and *Picricum acidum. Physostigma, Lachesis, Nitric acid, Rhus toxicodendron, Silicea* and *Graphites* have been credited with benefiting patients suffering from this disease.

In the writer's hands *Angustura vera* materially helped in one case where the lightning pains were of great intensity. *Carboneum sulphuratum* ought to be of signal service in restraining at least the advance of the disease, while *Zincum metallicum* and *Cadmium metallicum (vide* Erb's statement quoted in article on Syringomyelia), should be borne in mind. *Duboisia* and *Physostigma* both cause unsteadiness on standing or walking with eyes shut, but in the former it would appear from other symptoms to be vertiginous in origin, in the latter probably neuritic. Lippe (Repertory) gives *Nux moschata* the first rank as a remedy for tabes dorsalis, and the next and only remedy, *Phosphorus.*

From the foregoing it will be seen that in any case a close study of symptoms and especially of the ameliorations must be made; if we are to increase our therapeutic resources in the treatment of locomotor ataxia it must now be by recording all the symptoms and by collating the results of our prescriptions, and thus ascertain just in what respect and to what degree different remedies influence the course of the disease

The various crises, including the lightning pains, are

treated according to the symptoms of each case, especial-
ly the aggravations and ameliorations.   At the present
writing the pain crises, in a case in which the
only relief is obtained while the painful part is
immersed in water as hot as can be borne, have
been evidently influenced by *Strontiana carbonica* 30,
at frequent intervals during an attack.

Ataxic Paraplegia.

Whether the clinical picture seen in a case of
ataxic paraplegia is due to a distinct pathological
entity, or represents the results of separate disease-
processes in the lateral and posterior columns re-
spectively, cannot yet be decided.   Certain it is, that
cases having many of the distinctive symptoms of
spastic paraplegia, and many of those of locomotor
ataxia, do exist.   It is plain, however, that a com-
bination of the two diseases *per se* does not exist,
and, indeed, cannot exist, for some symptoms of
each are missing.

The disease is described as beginning usually be-
tween the thirtieth and fortieth years, but it has
developed as early as the fifteenth and as late as the
sixtieth year.   It is considered by Gowers to be an
isolated form of hereditary ataxia, but the writer has
under observation the case of a boy aged twelve, unable
to walk from the amount of paraparesis, the trouble
having begun at least as far back as his eighth year,
while he has double optic nerve atrophy (of the gray
or primary variety) with exaggeration of the knee

jerks, slight ankle clonus and hypertonicity of the
paretic lower limbs—symptoms that exclude Fried-
reich's disease absolutely.

The early symptoms are those of spastic paraparesis
with the addition of some ataxia. In the case men-
tioned above, the first symptom of ataxia was noticed
in the arms, the patient when trying to clap his hands
failing to succeed. Weakness of the legs, with too
ready fatigue, is seen, but the ataxia, present under
the usual tests and conditions, has more the quality
of reeling (when walking or standing) than that of
locomotor ataxia. Lightning pains and the girdle sen-
sation are present in rare cases and impairment of
sensation is likewise rare. The knee jerks are exag-
gerated, and usually ankle clonus can be elicited.
The bladder may be affected (retention paralysis)
while sexual power is frequently lost. Loss of the
reflex mobility of the pupil has been observed in
some cases, while optic nerve atrophy occurs at
times.

The disease advances chiefly in the direction of
increasing weakness of the lower limbs, with spas-
tic symptoms, when, of course, the ataxia is gradually
overshadowed, the patient, it may be, after a lapse of
years, becoming bed-ridden.

Some cases appear to be predominantly locomotor
ataxia, with an affection of the lateral columns super-
added.

The course of the disease is extremely slow,
death occurring after many years, and then usually

from some intercurrent affection, pyæmia from septic infection (bed-sores), or from nephritis, the result of long-lasting bladder trouble.

In the causation of the disease syphilis seems to have no part, while suppression of perspiration, excessive physical exertion, sexual excesses, etc., have been ascribed as immediate causes.

A similar clinical picture has followed the use of *Lathyrus cicera* (and *sativus*), and also the use of diseased corn (in Italy, under the name pellagra).

The treatment of ataxic paraplegia should be that of chronic myelitis, but from the facts named in the preceding paragraph, *Lathyrus cicera* or *sativus* ought to be of decided service; the writer has used it in diffuse myelitis with secondary degeneration with great benefit. *Ustilago* may be tried in view of a possible relation to pellagra, although our provings have shown its influence in the sexual sphere of women chiefly.

Hereditary Ataxia.—Friedreich's Disease.

This affection is one of the so-called "family" diseases, and appears in several members of the same generation. It is believed to be due to congenital fault, as often shown by the smallness of the cord, seen in autopsies of its victims.

The symptoms of the disease appear first about the time of puberty in most cases, although there has been reported as occurring as early as the sixth or seventh year a distinct ataxia of the lower limbs,

accompanied by some amount of muscular weakness. The gait is a modification of that of locomotor ataxia, since the weakness produces something of a "reeling" quality. Sensory disturbances are absent, lancinating pains having been only rarely noted, while hypæsthesia when observed has been only in the later stages. The knee jerk is lost early, as a rule. Choreiform movements occur in many cases, especially in the muscles of the neck, and then causing irregular oscillation of the head. The course of the disease is very slow, and several years may elapse before ataxia of the arms appears. Contracture in different muscles causes at times scoliosis, or talipes, or hyperextension of the toes, etc. Difficulty in speaking, the words being imperfectly enunciated, and nystagmus, are common features of the disease when it has well advanced. Paralysis of eyeball muscles, or loss of pupillary reflex, has been occasionally noted, but most observers insist that the eye fundus is always normal. Vesical troubles are extremely rare, and no "crises" occur in the disease.

As the disorder progresses, increasing weakness of the lower extremities eventually confines the patient to bed. It may extend over a period of thirty or forty years, but death comes from some intercurrent disorder, and, of course, at any time. The prognosis for cure is hopeless, but with care and proper hygienic regimen, life will be prolonged.

The anatomical changes, as shown by microsco-

pical examination are confined to the spinal cord and medulla chiefly, and the smallness of the cord seen in all cases examined is taken as evidence of imperfect development. The disease is really a combined system degeneration of the column of Goll entirely, the column of Burdach in part, the crossed pyramidal tract, the direct cerebellar tract, and in some instances the ascending antero-lateral tract. Changes in the medulla have not as yet been sufficiently well studied to deduce positive conclusions therefrom.

Spastic Paraplegia. — Spastic Spinal Paralysis.

This disease-manifestation was formerly called primary lateral sclerosis because of its clinical likeness to secondary degeneration (sclerosis) of the pyramidal tracts.

Its existence as an independent primary disease is now so stoutly contested that it must be a rare affection if it exist at all.

It is gradual in onset, weariness in the legs being first noted, advancing to paresis and later to paralysis, without sensory disturbance or trophic change, without implication of the bladder or rectum. A sense of stiffness is felt in the affected limbs, or a feeling as if the muscles were too short.

The existing hypertonicity is shown by the markedly exaggerated knee jerks and, in well advanced cases,

ankle clonus or even a patellar clonus (on pulling on the quadriceps femoris by means of the patella) may be observed. The affected limbs undergo contraction at times, from fatigue or in attempting a movement. The contractions are temporary at first, but in course of time a condition of rigidity comes on mostly of the extensor and adductor muscles. In consequence, the gait becomes the well-known spastic gait, the knees nearly together, the legs slightly bent at the knee; the steps taken are short and the balls of the feet scrape the ground, the heels not reaching it.

At each step the weight of the body causes an ankle clonus, thus giving a quivering quality to the gait.

The upper limbs may be affected in course of time with stiffness, impaired activity and increased deep reflexes.

The affection is slow in its progress usually; it may continue for years or may cease to advance.

Sections made from the cords of those who had the affection during life have not shown an isolated primary disease of the pyramidal tracts, but have shown other diseases or secondary degenerations with implication of the lateral columns. The trouble is now believed to be in most cases a result of syphilitic myelitis, diffuse myelitis, multiple sclerosis, tumor of the cord or brain, syringomyelia or even hydrocephalus.

Nevertheless, Oppenheim maintains the possibility of the existence of the independent primary affection, but in every case with these symptoms endeavor should be made to discover some cause such as those mentioned.

Hysteria is said to simulate the condition.

The diagnosis is made chiefly by determining the presence or absence of any primary affection.   In hysteria there may be a deceptive false clonus, but the onset of the symptoms is sudden and usually after some psychical excitement, while the rigidity has the peculiarity of hysterical contracture.

The symptoms of spastic paraplegia in the adult appear between the 20th and 40th years, as a rule. The cause assigned is at times trauma or exposure to cold ; a preceding syphilitic infection or an acute infectious disorder or even lead poisoning, is stated by Oppenheim to be capable of developing the disease.

The treatment should be that laid down in the section on chronic myelitis ; *Lathyrus sativus* or *cicera* has produced the symptoms.

Non-Systemic Diseases of Spinal Cord.

Multiple Sclerosis.—Disseminated or Insular Sclerosis.

A condition in which numerous small foci of sclerotic change are distributed widely and irregularly through the brain and spinal cord. In size these foci vary from that of a pin's head to that of a large bean, and, while they occur in the gray matter, are in greater numbers in the white. In accordance with their predominance in any special area or tract will the resulting symptoms differ, so that theoretically a case may occur which cannot be diagnosticated during life. Certain groups of symptoms, however, are so closely associated in the disease that most cases, as far as we can judge, can be diagnosticated. It is a disease seen mostly in early adult life, but cases have been reported in which it has appeared as late as the forty-fifth and as early as the fifteenth year. The writer has seen a case in which the tremor did not come on until the forty-seventh year, but the speech difficulty dated from early childhood.

The etiology of the affection is unknown, but its manifestations frequently follow the cessation of some infectious disease, such as smallpox, scarlatina, measles, etc., or appear as the effect of trauma or of violent emotion. Strümpell has lately advanced the hypothesis

that the disease is primarily a multiple gliomatosis, due to inborn conditions.

In a typical case the patient shows a certain interference with voluntary motion of the hands and arms, of the eye-balls and of the laryngeal muscles. The arms and hands, when the patient attempts any movement with them, are seized by a tremor, of small amplitude at first, but increasing in extent as the motion is continued. If told to take up a glass of water and drink from it, he shows a simple tremor on taking hold and the tremor becomes so wide in its increasing excursions that the contents of the glass may be violently thrown out. All the tremor ceases when the patient is at rest; but as the head and trunk muscles may be implicated they may continue in tremor when the patient is sitting or standing, because they are then in action. The eye-balls, when the gaze is directed to one side, show oscillation, known as nystagmus; often the nystagmus is present when the patient simply looks ahead. The speech difficulty has not been analyzed as to its muscular components, but is made up of somewhat jerky breaks in the emission of speech sounds, so that words are dissected into syllables and there is absence of the usual falling and rising inflection commonly heard in ordinary conversation. From these peculiarities the speech of insular sclerosis is termed "scanning" speech or syllabic utterance.

Besides the foregoing characteristic symptoms there are others of scarcely less diagnostic value. The

symptoms of spastic paraplegia are present in greater or less degree; weakness, with stiffness of the legs, spastic gait, exaggeration of the knee jerks.

Optic nerve atrophy is frequently seen in the course of the disease, more frequently than was formerly supposed. (Buzzard.) It appears first as paleness of the temporal half of the disk, but eventually involves the other half, yet not with such loss of visual power as might be supposed from the ophthalmoscopic appearances.

The mental state of the patient is altered; he is usually in a pleased or contented frame of mind, and fits of irrepressible laughter without cause, either objective or subjective, are not infrequent in the early stage; they have no relation to mental decay. (Oppenheim.)

Disturbances of sensibility, mostly paræsthesias of fingers and toes, appear in some patients, but are only temporary. Attacks of vertigo are found in the majority of the cases. During the course of the disease in some instances, apoplectiform attacks with perhaps complete loss of consciousness appear and are at times followed by a hemiplegia that soon disappears; such attacks materially injure the patient. Occasionally paresis of the extrusor or of the detrusor of the bladder occurs, but does not become permanent.

Aggregation of the islets of sclerosis or their increased size in some definite region will give rise to corresponding symptoms, such as atrophy, paralysis of bulbar nerves, or tabetic symptoms with loss of

the knee jerk (Gowers); but the Argyll-Robertson pupil does not occur, although inequality of pupils has been reported; lightning pains have been observed. Paralyses of some of the muscles of the eye-ball are not uncommon, and optic neuritis has been noted.

The disease may present a picture of transverse myelitis or of primary spastic paraplegia; the cerebral symptoms already mentioned will place the affection nosologically, even when the typical symptoms are absent; but dementia paralytica must be excluded.

Pathologically, the islets show increase of connective tissue with loss of the medullary sheaths of the nerve fibres, while the majority of the axis cylinders remain for a long time.

The development of the disease is slow, its course is very gradual; it may last ten or fifteen years, but it may remain stationary for long periods and actual remissions occur.

A neurosis has been described with many of the symptoms of multiple sclerosis, and hysteria has repeatedly exhibited intentional tremor. Nystagmus is present in other affections of the brain and also in independent ocular troubles, and intentional tremor has been observed in connection with cerebellar lesion.

The prognosis is absolutely bad as regards cure, but the possibility of cessation of advance or remission in the disease gives a certain hopefulness to the outlook in cases without threatening bulbar symptoms, or in those in which the apoplectic manifestations have not appeared.

Treatment. Claims have been made that the disease has been been benefited by the use of *Physostigma venenosa*, *Argentum nitricum*, *Plumbum*, *Nux vomica* and *Phosphorus*, but the writer has not been able to influence its course by any remedy. If Strümpell's hypothesis of the essential nature of the disease be well founded, it might be possible to retard its progress by our deep acting constitutional remedies such as *Sulphur*, *Calcarea*, *Lycopodium*, *Silicea*, *Thuja occidentalis*, etc.

Acute Ascending Spinal Paralysis.

Under this term Landry described a special form of disease in which, after some prodromal symptoms, general in character, a lax paralysis ensues, first in the legs and more in one than in the other in the beginning, with fever, and, in many cases, intestinal symptoms. The patient is compelled to remain in bed and the weakness attacks the muscles of the abdomen and then those of the arms. Next follow bulbar symptoms, consisting of difficulty in swallowing and in the use of the organs of speech. Attacks of dyspnœa, culminating in paralysis of respiration as the final term of the series, if continued, end the scene within a few days.

The ordinary sensibility is not disturbed, the tendon reflexes are at first not changed, but later become extinguished, and the functions of the bladder and rectum are, as a rule, not affected.

The electrical excitability of the muscles remains

undisturbed and there is no atrophy.   There are also
no mental symptoms.   Profuse sweating is generally
present, and some œdema may be observed.   The
fever may continue during the whole course of the
disease.   Enlargement of the spleen has been repeat-
edly noted.

The progress of the disease is rapid, in some cases
the fatal end occurring within two or three days,
or it may be delayed for a week or two.   Cures do
occur, but the convalescence is very slow.

From the foregoing it will be seen that the prog-
nosis is serious, and as all the cases reported do not
seem to be of one character, it is difficult to assign
any given case to a definite place in classification.
Some are undoubtedly instances of multiple neuritis,
and some appear to be cases of myelitis, but perhaps
the real character of the affection is that of acute
ascending infectious neuritis, with final implication of
the structures of the medulla and possibly of the cere-
brum.   From the symptoms, *Phosphorus*, *Oxalic acid*,
Lathyrus sativus, *Ledum palustre* and *Aluminium metal-
licum* would appear to be indicated.

The Caisson Disease.—Diver's Paralysis.

In laying the foundations of bridges or in other
work done at considerable depths below the surface
of the water, the workmen are in an atmosphere
whose pressure is two, three or, in some instances,
five times that of the ordinary atmosphere.

Under the pressure mentioned the men suffer but

little, if at all; indeed, there is a sense of stimulation, but if the pressure be rapidly lowered there come on in a short time some serious or even dangerous symptoms which are relieved if the patient can be returned to the pressure chamber. If the pressure be gradually reduced to that of the ordinary atmosphere no ill effects follow, and an arrangement of chambers with successively decreasing pressure has been in use, through which the workmen pass into the outside air, staying for a time in each; or, lately, one intermediate chamber only, with a means of slow escape from it of the compressed air, serves even better the desired end.

When through the recklessness of the workman or insufficiency in the method of lowering the pressure, the change from the working pressure to that of the outside air is made too rapidly, the symptoms of the disorder come on.

Under the influence of increased atmospheric pressure there is taken up through the lungs a proportionally increased amount of the constituents of the inspired air, in accordance with the physical law of absorption of gases by liquids. If the pressure is gradually lessened the absorbed gases are gradually released from the circulation, through the lungs and without damage; if the pressure is rapidly lessened, the absorbed gases appear as bubbles within the containing liquid. In the case of the larger blood vessels, these are passed along in the circulation, but in the case of small vessels,

and especially the terminal arteries, the minute
bubbles act as emboli, occlude such vessels, and cut
off the blood supply to the area of tissue supplied
by them. According to Kadyi, the arteries supply-
ing the lateral and posterior aspects of the cord
are smaller than those of the anterior and central
portions, and in the whole dorsal extent of the
cord the vessels are especially small. Consequently
this part becomes the site by predilection for the
gas emboli, with consequent spinal symptoms and
secondary degenerative changes in the cord. The
liberation of the gas in minute bubbles outside of
the blood vessels, or through them and the pos-
sible production thereby of minute fissures in the
cord, are of much less importance than the areas
of necrosis, softening and reactionary inflammation
set up in the cord in the manner stated. A full
discussion of the subject is given in a paper by
Dr. A. Hoche, of Strasburg, in Berliner klinische
Wochenschrift, 22, 1897.

The symptoms are felt in from two to thirty
minutes after leaving the pressure chamber too
rapidly, some time being required for the evolution
of gas bubbles.

Nitrogen is the chief constituent of the gas bub-
bles, it not being so readily held by the blood as
oxygen or CO_2.

The symptoms are evidently of spinal and cere-
bral origin: vertigo, pain and noises in the ears,
nausea, and even vomiting, with weakness in the

legs. Intense neuralgic pains in the muscles and joints of back and lower limbs, with anæsthesia of the surface of the legs, and often with retention of urine. Slight cases recover in a few days or a few weeks; severe ones may die early, or after lingering for some months. Permanent damage to the cord, with secondary degeneration, is often the sequel, as shown by paraplegia, paresis of bladder, etc.

The greater the pressure and the longer the time the worker remains in the working chamber, the greater is the amount of gas taken up by the blood; hence the need of gradually lessening the pressure as the workman passes out into the ordinary atmosphere.

As soon as symptoms appear, the patient should be returned to the pressure chamber until relieved, and then slowly emerge into the open air through the graded pressure chambers. Even up to the third day this should be done, for P. Bert in his experiments on the effects of increased atmospheric pressure on animals, found on the fourth day after symptoms had developed, gas emboli and blood coagula within the vessels of the spinal cord.

Otherwise the treatment by internal remedies should be symptomatic. *Arnica* and *Rhus toxicodendron* would appear to be the best suited to the early symptoms; later, the case may be treated as one of chronic myelitis.

Tumors of the Spinal Cord.

In the section on Compression Myelitis, tumor within the spinal canal is mentioned as a cause of the symptoms, yet the subject of tumor within the canal needs separate consideration.

Tumor affecting the spinal cord is most often meningeal in origin, less frequently it arises within the cord itself, and rarely from the vertebræ. It may, therefore, be between the dura and the bony wall of the spinal canal, or enclosed within the membranes, or between the membrane and the cord itself, or wholly within the latter.

In the first three cases the effect of a tumor is to produce compression of the cord, often with great irritation of the nerve roots, with symptoms resulting therefrom; in the fourth case the symptoms will be those of diffuse myelitis, at times with peculiar dissociation in the qualities of peripheral sensibility, as in syringomyelia (q. v.).

Tumors arising from the meninges are sarcomata, myxomata, enchondromata and lipomata; those occurring in the cord itself are gliomata, sarcomata, gummata and tubercle.

Tumor of the cord is generally small, but glioma and sarcoma may involve many segments of the cord. Solitary tubercle may be as large as a hazel-nut.

The symptoms of tumor of the spinal cord already mentioned become peculiar in their distribution when only one lateral half of the cord is affected. The result then is Brown-Séquard's paralysis, in which

there is motor paralysis and absence of the muscular sense below the site of the tumor and on the same side, while sensory paralyses, chiefly of the pain and temperature senses, are found in the corresponding parts of the opposite side. At the level of the lesion a band of anæsthesia on the side of the lesion, and one of hyperæsthesia in the corresponding part of the opposite side exists.

With meningeal tumors the irritation of the nerve roots causes severe pain in the distribution of the nerves so irritated, and such pain may long precede the symptoms of compression of the cord.

From irritation of the nerve roots, spasmodic contractures of the limbs are observed; they may be very severe.

The reflexes are increased when the lateral columns are affected, except when the lesion is situated in the lumbar enlargement of the cord, and then the knee jerks are absent.

Paræsthesias, hypæsthesia, or anæsthesia may exist.

Paralysis comes on in the course of time; it may be of one or of all four extremities, according to the site and lateral extension of the tumor; some atrophy may be present.

Death follows from exhaustion, or sepsis, preceded by paresis of bladder and rectum and formation of bed-sores in a considerable proportion of cases.

The pathological changes resulting from tumor of the cord are flattening, and decrease in its size in cross-section, with marked softening, studded with

small hæmorrhages. Secondary degeneration and re-active inflammation serve to complicate the symptoms of the case.

The diagnosis is to be made from Pott's disease, transverse myelitis and pachymeningitis cervicalis hypertrophica. In the first, the age of the patient, the presence of tuberculous trouble elsewhere, and the marked stiffness and painfulness of the vertebral column, together with angular projection of some of the spines, are decisive. In the case of myelitis there is not the gradual increase of the symptoms, nor the succession of them found in tumor of the cord. At the same time it must be held in mind that a tumor wholly within the cord will usually not give rise to the severe neuralgic pains found when the neoplasm is outside of it. Pachymeningitis is to all intents and purposes an extra-myelonic tumor.

The character of the tumor may be inferred if in any other part of the body a tumor exist; gumma may be inferred from antecedent history of syphilitic infection.

Tumors arising from the membranes show but little tendency to invade the structures of the cord, and the latter exhibit a remarkable resisting power to the increasing pressure (A. Westphal).

The prognosis of tumor within the spinal canal is absolutely bad, but surgical interference has occasion-ally rescued the patient from his threatening fate. In a study of 123 cases, of which 100 were described in detail, by M. Allen Starr, 22 cases were operated on, yet in only 6 were the patients relieved of the

paralytic symptoms, probably because surgical inter-
ference was at too late a period. Starr states that
most intraspinal tumors are malignant, and that al-
though this darkens the prognosis, still operation
should not be refused, provided the tumor is extra-
myelonic, which fact is difficult of ascertainment. He
considers as evidences of intra-myelonic tumor, Brown-
Séquard paralysis, early degenerative atrophy, trophic
changes and bedsores, and the appearance of analgesia
before anæsthesia.

The medicinal treatment of intraspinal tumor can-
not be on the diagnostic indications, for the tumor
is simply a foreign body within the spinal canal,
irritating and compressing nerve roots and cord
structures. The symptoms thus produced are not
those of the disease itself. Hence, if prescribing
our remedies is to be of service they must be chosen
according to the symptoms of the patient's person-
ality, the aggravations, ameliorations, etc. If the diag-
nosis of tubercle or of syphiloma can be fairly as-
sumed, the isopathic method may be of value, and
in the latter instance the use of the remedies sug-
gested in the section on syphilis of the spinal cord
is recommended.

Syringomyelia.

Proliferation of glia cells and fibres in the central
gray of the cord results in the formation of a tumor,
a glioma. Increasing growth compresses the blood
supply at its periphery and the centre breaks down,

forming a cavity more or less tubular, and to this condition the term syringomyelia is applied.

As the central gray is invaded and in part destroyed the symptoms are peculiar.

The cause of the trouble is primarily an inborn defect or morbidity of the affected region itself, but often some exciting cause (trauma) is needed to start the process into activity.

The symptoms of the disease manifest themselves most often in late youth or early middle life. In one or both hands a slowly progressive muscular atrophy appears, perhaps preceded by some pain, vague in character. The atrophy is similar to that observed in progressive muscular atrophy of spinal type and, like it, it may first appear in the muscles above the elbow or around the shoulder. Fibrillary contractions may be noticed in the atrophying muscles, whose electrical contractility is lessened or changed at times to the reaction of degeneration. The grouping of the muscles affected depends upon the direction in which the gliosis advances in the cord. Most frequently it involves the cervical enlargement, often implicates the dorsal segments, rarely the lumbar portion.

The special and characteristic clinical feature of the disease is the peculiar anæsthesia for certain qualities of sensation. In the affected member and over a larger area than that of the atrophy, there is loss, often complete, of sensation for pain and for temperature, so that a needle prick or the faradic current is not appreciated as such, and hot or cold

bodies when applied to the anæsthetic region cannot
be differentiated from simple touch, tactile sensation
being retained. The muscular sense is not impaired.

Paræsthesias, a feeling of cold or of heat or a mix-
ture of the two, are present in most cases, but sub-
jective pain is rare.

Trophic and vaso-motor disturbances are frequent.
Blebs and bullæ may form upon the skin of the
affected parts and, bursting, leave ulcers often difficult
to heal. Eruptions, eczematous or herpetic, are not
infrequent, while bluish-redness or œdema of hands
or arms has been observed. Panaritia with anæsthesia
and analgesia (Morvan's disease) has been so often
associated with syringomyelia that it is held by many
to be a manifestation of the disease; others, however,
maintain that it is due to associated neuritis. Scoli-
osis and kypho-scoliosis may appear in the course of
the affection. From pressure on the lateral pyramidal
tracts there may follow at a somewhat advanced period
spastic paraparesis.

The gliosis may predominate in one-half of the
cord, when the symptoms will be practically unilateral;
it may extend in more than one direction or have
more than one cavity; it may extend upward, involv-
ing the medulla, and so give rise to bulbar symptoms;
if it extend low down in the cord, the legs will be
affected in the same way as the arms.

Inequality of pupils is not rare, and concentric
narrowing of the visual fields often exists. (Gold-
scheider.)

Diagnosis. In a typical case the peculiar sensory disturbance with the associated atrophy will be sufficient for the diagnosis. But not all cases are typical and when the sensory symptoms are absent it will be impossible to decide between this disease and progressive muscular atrophy of spinal type or amyotrophic lateral sclerosis.

Hydromyelia. By this term is meant a congenital condition analogous to and at times associated with hydrocephalus, in which the central canal is widened for a greater or less extent, usually more posteriorly than anteriorly, and at times irregularly. Unless pathological changes occur in its walls and adjoining tissues, it has no clinical importance.

The treatment of syringomyelia can only be symptomatic and then not guided so much by the diagnostic symptoms as by those that characterize the patient, such as his aggravations and ameliorations or peculiar symptoms not part of the special trouble.

It is interesting to note a report of two cases having all the symptoms of syringomyelia which appeared after " running " a chip or shaving of zinc into a finger and a thumb, respectively, reported by Dr. Joseph Mies of Cologne (Münch. med. Wochenschrift, 19, 1896). The men were both healthy at the time of the injury, one aged 40, the other 33. The symptoms were gradual in development; in one case the diagnosis of progressive (spinal) muscular atrophy was made after some four years, the temperature and the pain sense not being then affected.

After eight or nine years the temperature and pain senses were found affected. The other case was more rapid in its progress, but the results were the same. The reporter considered the symptoms to be the result of slow poisoning by zinc, and compares the cases to those reported by Schlockow as cases of peculiar spinal cord affections in workers in zinc.

Eulenburg, in commenting on Mies' cases, rejects the theory of chronic poisoning by zinc, and considers an ascending neuritis to be the middle link between the peripheral injury and the consecutive spinal cord affection. (Deutsche med. Wochenschrift, 29, 1896.) He further considered that Schlockow's cases of various troubles, due to inhaling the vapors of zinc, were chargeable to impurities present, especially arsenic, cadmium and lead.

It may be worth noting that in both of Mies' cases the splinter or shaving of zinc had been touched with ordinary hydrochloric acid, which may have made in the wound zinc chloride, a soluble poisonous compound.

Treatment. Since the affection is an invasion of the cord by the proliferation of glia cells, the symptoms will vary according to the parts of the cord affected either destructively or by pressure. To prescribe on such indications cannot be expected to influence the advance of the real disease. Hence, the diagnostic symptoms should not be taken into account in searching for a remedy, but reliance placed on the symptoms of the patient that are constitu-

tional, the times and conditions of aggravation, amelioration, etc. The possibility of special influence of the metals mentioned in causing organic change within the cord should be held in mind in prescribing for the disease.

The manifestation termed Morvan's disease, whatever be its cause, would lead us to use remedies known to be of especial service in panaritia, such as *Dioscorea villosa*, *Natrum sulphuricum*, *Silicea*, *Sulphur*, *Hepar sulphuris calc.*, etc., but analgesic panaritium is a special manifestation not as yet noted in the provings.

Spinal Irritation.

Under this name a set of symptoms referable to the posterior or sensory spinal nerve roots, is frequently found in hysteria, neurasthenia, or may exist alone.

The symptoms are pain in the back with tenderness to pressure or at times even to touch and often with a subjective sensation of burning. The patients complain of tiredness on exertion and the exertion is apt to increase the painful sensations in the spine.

Painful pressure points may be found at the sides of the vertebræ, about the lower cervical and upper dorsal and in the lower dorsal and upper lumbar regions. Pressure may result in simple increase of the already existing pain, in the production of an added sensation of soreness *in situ*, or in causing pain or other annoying sensation in the limbs or other distant parts.

When the seat of the trouble is in the lower cervical or upper dorsal region, the use of the arms, as in holding a book, in sewing, or in doing up the hair (in women) aggravates the condition. In one case the midcervical region seemed most affected, and then the pressure of the coat collar was intolerable. If the dorsal region is affected, the patient may be unable to sit with the back resting against the back of a chair.

Symptoms of neurasthenia or hysteria are often present, and are given under those heads.

The affection appears in adolescence and in early adult life, and, as it is often slow in onset, adequate cause may not be discoverable; the general terms overwork, worry, overstudy, etc., may mean much or little in this connection. The writer has seen the affection in a number of cases follow an attack of grippe. As cases have been cured by relieving uncorrected errors of refraction or other ocular troubles, intrapelvic disorders, etc., it has been assumed that such conditions are etiological factors in producing the affection. Sexual over-indulgence, whether in a normal or abnormal manner, is a causal factor in many cases.

The lowered vascular tonus, as evidenced by relaxed blood-vessels of face and hands in many cases, is probably existing within and will account for the spinal symptoms; engorgement, especially of the veins within the spinal canal, results in pressure and irritation of the nerve roots, particularly of the posterior or sensory ones; and most of the associated symptoms,

such as weak digestive power, constipation or diar-rhœa, palpitations, etc., may be thus accounted for, either directly or indirectly.

The knee jerks are generally exaggerated.

The diagnosis is to be made between this affection and an organic disease, such as compression myelitis, spinal meningitis or meningo-myelitis. In organic af-fections there is motor weakness, often with rigidity, the weakness may amount to paresis or paralysis, or the pains radiate from the spine instead of being limited to it. The history, the continuance of the symptoms for long periods without much change, the absence of paralysis of bladder or rectum, are in favor of spinal irritation.

The prognosis for cure is good.

Treatment should be both medicinal and hygienic; it will be given in the section on neurasthenia.

The Cranial Nerves.

During the last few years the effort has been made (and with a large measure of success) to apply the rule of uniformity to the origin of both the cranial and the spinal nerves. The latter, as has been stated in the section on the spinal cord, arise in two ways: the motor nerve fibres from large ganglion cells in the anterior gray horns, the sensory fibres from the posterior spinal ganglia, whose cells give off fibres that at a short distance divide, one part going to the periphery and ending there in free division or in a specially developed end organ, the other part passing into the region of the posterior gray horn of the cord.

Of the cranial nerves, those having motor function arise from large cells aggregated in masses within the brain structure, such masses being analogues of the cell-groups in the anterior gray horns of the cord.

Similarly, some of the cranial nerves subserving sensation, notably the auditory, and part of the fifth, arise from ganglion cells outside the brain structures proper.

In the case of the olfactory and visual functions the analogy is not so close.

On the base of the brain is seen anteriorly on each side, lying in a groove or sulcus at the bottom of the frontal lobe, the so-called **olfactory nerve,** whose anterior end is somewhat enlarged into the olfactory

bulb, which is the residue of an enormous development of brain structure in animals endowed with the olfactory sense in a high degree.

The olfactory bulb contains specialized globular structures known as glomeruli, granular cells and star cells and giant ganglion or "mitral" cells.

Buried in the mucous membrane of the upper and inner nasal cavities, besides the epithelial cells proper, are specialized olfactory cells from which there pass backwards fibres through the cribriform plate which then almost immediately end in a brush work that half surrounds a glomerulus in each case on entering the olfactory bulb.

The opposite half of the glomerulus is enclosed by the brushy endings of the dendrites of a mitral cell.

It would therefore appear that the peripheral neurones of the *fila olfactoria* form the real olfactory nerve and that the fibres going brainwards from the mitral cells (secondary neurones) form an intermediate tract which at the anterior perforated space divides into three so-called roots. The outermost is the largest and its fibres end in the cortex of the hippocampus; the middle division is poorly developed in man; the fibres of the internal division pass through the anterior commissure to the cortical area of the other side.

Loss of the power of smell is most frequently due to disease of the mucous membrane affecting the included olfactory cells and nerve fibres, to nasal polypus, to dryness of the nasal mucous membrane due to trophic changes following paralysis of the fifth nerve,

or to obstruction in the lachrymal canaliculi. Falls and blows upon the head may cause laceration and separation of the filaments of the nerve proper from their connection with the cells of the olfactory bulb. Disease in the anterior fossa, such as tumor, meningitis, bone affection, may cause compression of the olfactory bulb and tract. Excessive olfactory stimulation has caused loss of the faculty (Gowers) and senile atrophy of the bulbs has occurred in old age.

With the loss of the sense of smell, the sense of taste is usually lost for "flavors," the latter being appreciated *via* the posterior nares. Loss of the former with retention of the latter is due to disease of the mucous membrane in the upper part of the nasal passages.

Anosmia, due to cortical disease or to disease back of the internal capsule, cannot be diagnosticated by the olfactory symptoms alone. The combination of aphasia and right-sided hemiplegia with left-sided anosmia has been observed several times; it is probably due to implication of the external olfactory root, near the island of Reil.

Functional anosmia is seen at times in hysteria and neurasthenia; in the former it may be part of the hysterical anæsthesia.

Congenital absence of the olfactory tracts and bulbs has been met with.

Hyperosmia or increased sensitiveness of the olfactory nerves is seen at times in the hysterical and the insane.

Hallucinations of smell occur at times in the insane, and occasionally as the aura preceding an epileptic spasm.

Parosmia, or perversion of the sense of smell, is a rare condition, in which every impression made upon the olfactory nerve is translated, so to say, as a disagreeable odor, always the same. The condition is probably cortical.

Treatment must be influenced by the diagnosis of the underlying condition, and hence examination of the nasal passages, as well as a knowledge of the history, is imperative. If local conditions, past or present, place the affection in the category of purely nervous trouble, the matter of treatment becomes that of the cause. In the absence of such condition other symptoms must be relied on for making a diagnosis of causes within the cranium; if neither these nor intra-nasal disease be present, the treatment must be purely symptomatic.

The prognosis in the nervous forms, whether of the bulb, tract or cortex, is not encouraging.

The optic nerve is similar in its beginning to the olfactory. The rods and cones of the retina are the specialized sensory end organs of vision. The former terminate in prolongations, with knobbed ends, the latter divide each, and then give off short branchings. So-called horizontal cells connect functionally the endings of the neighboring groups of rods, while another set of cells carry impulses from

rods and cones to large ganglion cells, from which arise the long nerve fibres that pass brainward. If we push our analogy, we may say that the rods and cones, with their endings, form the real optic nerve.

All the fibres of what has hitherto been understood as the optic nerve are not centripetal in their course. There are centrifugal ones, probably from cells of the anterior corpora quadrigemina.

The optic nerve, as we must continue to designate the long nerve fibres, passes backward and toward the middle line, where a partial decussation with the nerve of the other side takes place, and in such manner that the fibres from the inner halves of the two retinas cross, those from the outer half of each retina continuing on their own side.

Behind the crossing, or chiasm, the mass of fibres is termed the optic tract. It winds around the crus of its own side and passes upward, giving most of its fibres to the external geniculate body, a few only to the anterior corpus quadrigeminum of the same side. From cells of the external geniculate body new fibres arise, which pass backward as the optic radiations of Gratiolet, and end in the cortex of the cuneus and its continuation on the posterior aspect of the first occipital convolution.

In the cortex of the occipital lobe both retinas are represented, the light-impulses from the outer half of the retina of its own side, and those from the inner half of the opposite retina being brought together. Fig. 22, from Bechterew, gives a diagram-

matic representation of the course of the fibres, and explains also the production of hemianopsia by a lesion affecting the fibres anywhere behind the chiasm.

Fig. 22. From Bechterew. The course of the light-impulses from their starting point in the retina to their arrival in the cortex (cuneus) is shown. The fibres, from the inner half of each retina, are seen to cross at the chiasm *ch*; the optic tract ends in the external geniculate body *nge*, and the anterior corpus quadrigeminum of the same side, *qa*; some fibres may end in the posterior mass of the optic thalamus, *p, th*. New fibres arising from these structures form the optic radiations *trv*, and end in the cuneus *C*. The commisure of Meynert, *CM*, and that of Gudden, *cg*, have nothing to do with carrying light impulses, but connect the large basal ganglia of the two hemispheres, probably.

The retina is designated by the part marked *r*. The pupil is not represented, but it is readily understood that rays of light from the right must impinge on the left half of the retina. A lesion at *no* is seen to affect all the fibres from the retina of the left eye; hence no light impulses can pass back, and that eye is totally blind. A lesion in the optic tract, at *tro* or at *l*, is seen to affect only the fibres coming from the left half of each retina. Hence each eye is half-blind and in the same-named half of each retina. This hemianopsia takes its qualifying word not from the half of each retina, but from the absent half of the visual field. In the case assumed the lesion is in the *left* optic tract or in the *left* optic radiations, and there must exist loss of vision in the *right half* of *the field*—that is, a right hemianopsia.

Fig. 22.

A lesion at *ch* will affect the decussating fibres only, and there will result a bitemporal hemianopsia; while to produce a bi-nasal hemianopsia there must be two lesions one on each side of the chiasm.
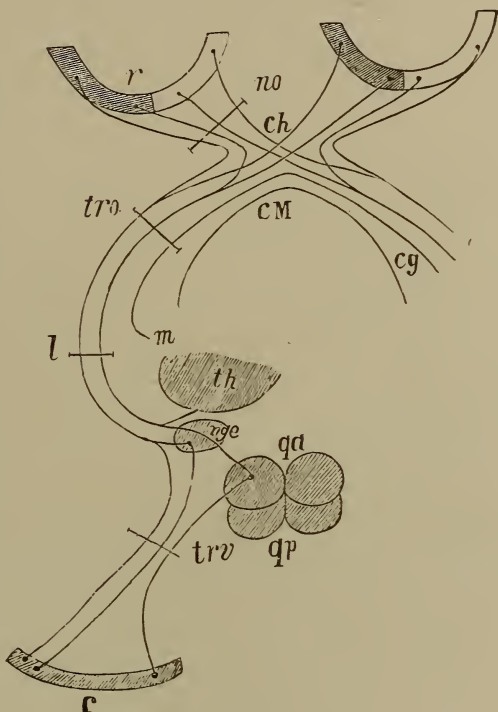
The optic nerve is the only nerve that is accessible to inspection; here, by the aid of the ophthalmoscope, the retinal expansion of the nerve can be studied in health and disease, and alteration of function or structure is, speaking generally, as much the subject of neurology as of ophthalmology. Strictly speaking, diseases of the retina itself, such as retinitis albuminurica, retinitis pigmentosa, or choroido-

retinitis syphilitica belong to the latter department of medicine, yet they must be recognizable by the student of the former.

On the other hand, the atrophies seen in the disk, whether primary (in association with degenerative disease of the spinal cord or brain), secondary (resulting from injury to the nerve at or in front of the chiasm) or consecutive (to optic neuritis), are of the highest interest to the student of nervous diseases.

Testing the visual acuteness, the color sense and the visual field is often a necessary part of the examination in nervous disease, since the results thus obtained may be decisively diagnostic or of high corroborative value.
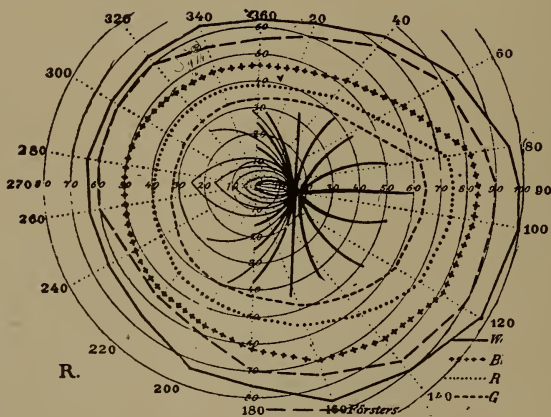
Fig. 23.

Fig. 23   From Baas.   Outlines of the visual field of the right eye, lessening of whose outline is abnormal (Förster's minimal visual field). The fields for colors are indicated by the different methods of outlining, the key to which is at the lower right of the figure.
   The lines gathering together in the centre indicate the coming together of fibres from different parts of the retina and meeting to form the optic nerve at the disk, beneath which they assume their medullary sheaths.

The pupillary reflex must be tested both directly and consensually.   In the former, if no light-impulses

are sent back to the primary optic centre, no reflex contraction of the pupil can occur, but if light entering the other eye affects the pupil of the first one, we know that the third nerve of the first is not at fault and that the trouble is loss of conduction in the optic nerve of that eye.

So in hemianopsia we may obtain valuable information by the hemiopic pupillary test as to the site of the lesion. If by means of a plane mirror light be thrown on the half of the retina now rendered useless by the lesion, when the lesion is not further back than the corpora quadrigemina, the pupil will not respond; if the lesion is in the occipital cortex, or in the optic radiations between the cortex and the corpora quadrigemina (and external geniculate body, optic thalamus), the pupil will respond. The test is, however, difficult of application, but, theoretically, its value is great.

Loss or lessening of visual power often bears no relation to the evident change in the disk or head of the nerve. In papillitis, or so-called choked disk, the swelling may be very great, yet loss of vision be slight, and *vice versa* (*vide* section on brain tumor). On the other hand, loss of visual power may be due to error of refraction, and disappear as soon as the error is corrected by proper lenses. Hence the student of this branch of medicine should possess and be able to use a case of trial lenses.

Mapping the field of vision often tells a great deal. Central scotoma means loss of conduction of the

fibres occupying the middle of the nerve trunk be-
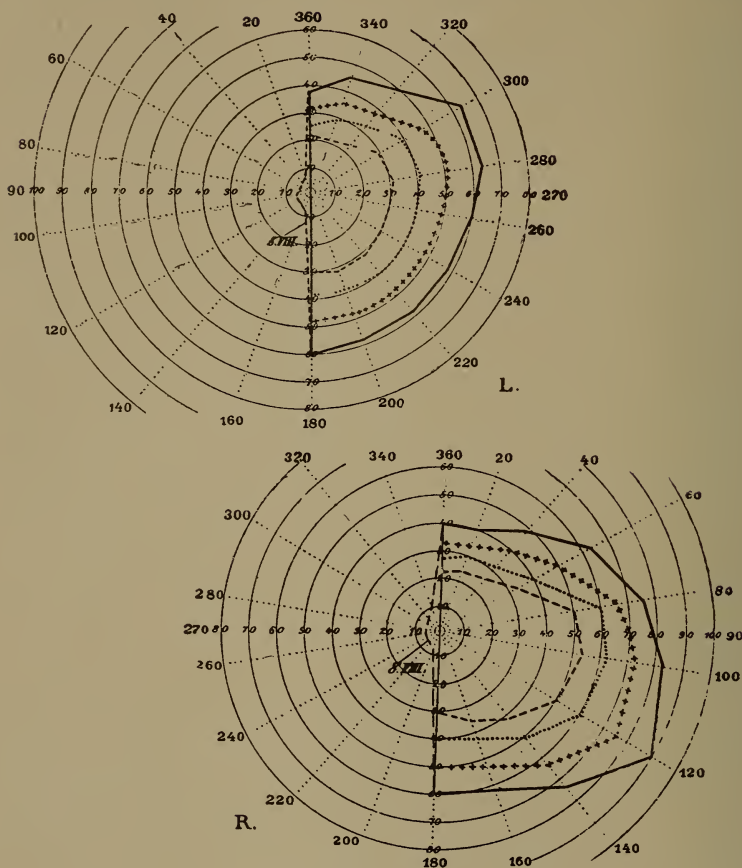hind the orbit; loss in the periphery of the field

Fig. 24.

Fig. 24. From Baas. The visual fields of a case of left homonymous hemianopsia. The
dividing line between the seeing and non-seeing halves, is seen to be perpendicular, but without
curving around the centre or fixation point, as it usually does. In this instance, however, the field
for green is seen to extend beyond the centre.

may mean an affection of the fibres at the outside
of the nerve trunk, or simply a lessening of con-
duction in all the fibres, normal vision being less
acute at the periphery of the field.

Changes in the color sense are to be expected

in optic nerve atrophy, and often precede ophthalmoscopic evidences, or even material contraction of the field for white. Congenital absence of the color sense (by no means a rare occurrence) will render nugatory the tests with candle and colored glass in diplopia.; in such cases the test must be by the use of prisms.

For details in the method of examination in the foregoing the student is referred to works on ophthalmology.

Optic neuritis or papillitis is an inflammation of the head of the optic nerve or disk. It is characterized by swelling, œdematous and inflammatory, lessened transparency of the tissues, with extension of the swelling to some distance beyond the limits of the disk. The edge of the disk loses its sharpness, becomes softened or "woolly" in outline, and
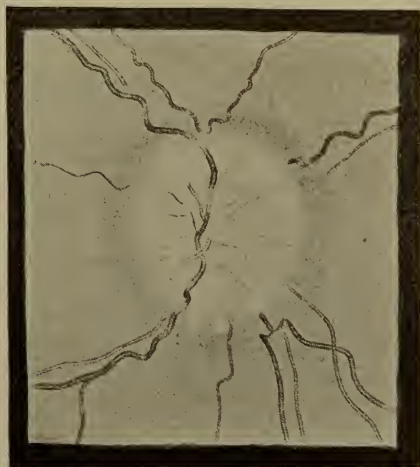
Fig. 25.
Ophthalmoscopic appearance of choked disk. From Gowers.

over the steep edge the vessels can be seen to descend. The veins are swollen and tortuous, the arteries narrow, and both disappear in places, being covered by the swelling. The general tint of the swollen papilla is reddish and striated. The term " choked disk " is given to the condition, but the term is misleading as implying a definite mode of origin, which, indeed, is now admitted to be not due to constriction or to pressure of fluid within the sheath of the nerve.

Neuro-retinitis (descendens) is believed to originate in the nerve or centres behind, but its ophthalmoscopic appearances differ from those of papillitis in being without any considerable swelling of the disk, and the uniform redness of the disk passing unchanged into that of the retina. Papillitis has swelling of at least ⅔ mm. equalling 2 dioptres of refraction.

The most frequent cause of papillitis is brain tumor; it also occurs with meningitis, abscess of the brain, sinus thrombosis, and occasionally in hydrocephalus internus, after acute infectious diseases, and rarely during amenorrhœa and anæmia.

The trouble is generally bilateral, but occasionally it is unilateral even when of central origin; unilateral optic neuritis is most frequently due to disease of the nerve at the back of the orbit, or at the optic foramen.

Prognosis of papillitis is largely a matter of diagnosis of its cause. When due to anæmia or amenor-

rhœa, the chances of cure are fair, especially as in these cases it advances rapidly and is apparently not accompanied by the destructive changes in the nerve which occur in the slowly advancing degeneration set up by brain disease. In any case, remedies must be addressed to the whole condition.

Papillitis, if unchecked, advances to consecutive atrophy of the nerve. This consecutive atrophy does not differ in appearance from that known as secondary atrophy, due to compression of the nerve behind the bulbus. In both cases the disks are usually of the brilliant whiteness of tendon; with the former, however, there may be white spots outside the disk in the region of the macula.

Primary atrophy of the optic nerve is seen in association with central degenerative disease of the nervous system, such as locomotor ataxia or dementia paralytica. Here the disk is generally of a bluish-gray or at times of a light muddy tinge. The atrophy accompanying multiple sclerosis is most marked in the temporal halves of the disks and is generally white.

Toxic influences, such as alcohol, tobacco, lead, bisulphide of carbon, mercury and (recently reported) *Filix mas* have caused atrophy of the nerve, in some instances preceded by papillitis. Such cases belong to the ophthalmologist, but are mentioned here as offering suggestions in the selection of remedies. The writer has used *Carboneum sulphuratum* 6, and fluid extract of *Filix mas* in 6th dilution in the hope of arresting the primary atrophy seen in lo-

comotor ataxia and dementia paralytica, but without result.

Functional amblyopia occurs in hysteria and in attacks of migraine, with concussion of the brain and after profuse hæmorrhage.

Temporary hemianopsia may be present in migraine.

Irritable retina is found in neurasthenia, hysteria, from excessive use of tobacco; it may follow exposure to very bright light or the reverse. The use of smoked glasses to protect the eyes is unadvisable under ordinary circumstances, the retinal hyperæsthesia being thereby increased.

The treatment of functional amaurosis or amblyopia must be largely that of the underlying neurasthenic or hysterical condition, or according to the cause, if any can be found.

At the New York Ophthalmic Hospital the remedies most used for neuro-retinitis are *Belladonna, Kalmia, Macrotin, Nux vomica, Phosphorus, Plumbum metallicum* and *Pulsatilla*—all in low dilution, generally 3x. *Duboisia, Mercurius corrosivus* and *Veratrum album* have been used less often. *Pulsatilla* has been found to be especially valuable in choked disk.

In Dr. Rounds' Clinic at the above named institution a case of optic nerve atrophy from abuse of tobacco and alcohol, without perception of light, has been so far improved under the action of *Agaricus* — 15 drops of the tincture three times a day — in 18 months that he is now able to walk the streets alone. A nephew and a son of this patient had the

same trouble, but refusing or being unable to stop using alcohol and tobacco have not improved. In all three cases the disk was brilliantly white.

The third nerve. This nerve, unfortunately termed oculo-motorius, arises from groups of cells placed beneath the floor and somewhat to the side of the aqueduct of Sylvius. The fibres pass downward and coming together emerge from the inner face of the crus in front of the pons.

From the occurrence of isolated palsies of eyeball muscles, experiments on animals and post-mortem examination (microscopical), it is held that each eyeball muscle is innervated by fibres from a definite group of cells. The arrangement of the groups as given by M. Allen Starr is the following:

Sphincter iridis	Ciliary muscle	
Levator palpebræ	Internal rectus	Median line.
Superior rectus	Inferior rectus	
	Inferior oblique	

In the middle line beneath the aqueduct is a central group of cells, while from the so-called posterior nucleus fibres are given off that pass into the nerve of the opposite side. This decussation is believed to concern the internal recti, whose action in convergence is distinct from the isolated action of one internal rectus.

The fourth nerve arises from a group of cells posterior to the third nerve nucleus. The fibres pass backward and upward to emerge as a nerve trunk behind the posterior quadrigemina and cross over the

middle line to wind around the opposite crus and appear at the base.

The sixth nerve which, for convenience sake, had better be considered here, arises from the cells of its nucleus situated beneath the floor of the fourth ventricle. The fibres pass downward and emerge next to the middle line and behind the posterior border of the pons.

The muscles supplied by the third nerve are already given in the table. The fourth nerve supplies the superior oblique, the sixth the external rectus.

The actions of the eyeball muscles are indicated in most of them by their names; the internal rectus turns the eyeball inward or toward the nose, the external rectus turns it outward or towards the temple, the superior rectus turns it upward; the inferior rectus turns it downward; the oblique muscles act similarly to their opposite named recti—thus the superior oblique turns the eyeball downward, the inferior oblique turns it upward, while both obliques turn it somewhat outward, and both superior and inferior recti turn it somewhat inward.

In turning both eyes to the right or to the left there must be an associated action of the internal rectus of one eye with the external rectus of the other. Cases occur in which with a paresis of the external rectus of one side the internal rectus of the other side is inactive in binocular vision, but if the first be covered the internal rectus of the second acts perfectly. From this it is inferred that the sixth
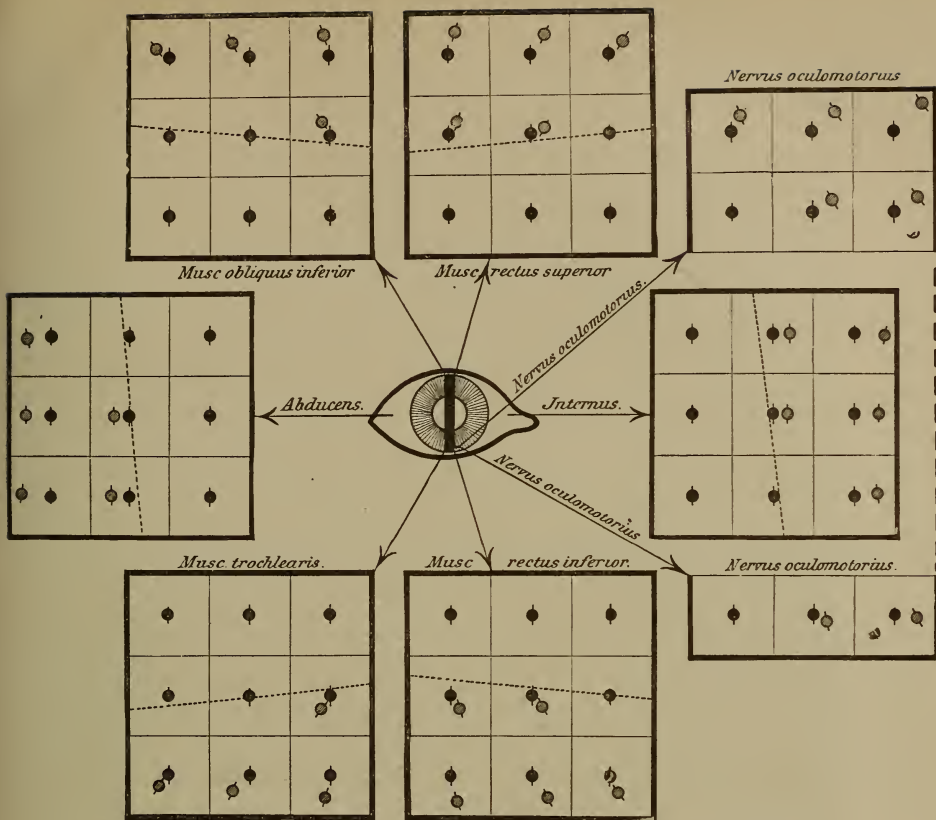
Fig. 26.

Fig. 26. From Roth. In the illustration of diplopias resulting from paralysis of different muscles of one eye, each large square represents the binocular visual field, subdivided as follows :

UPPER OUTER	UPPER	UPPER INNER
OUTER	DIRECTLY IN FRONT	INNER
LOWER OUTER	LOWER	LOWER INNER

The reader sees the double images in the drawing as if his own left eye were affected. To make the drawing represent paralysis of a muscle of the right eye he must represent himself as being in front of the patient and mentally view the double images from that position.

The perpendicular meridian of the cornea is indicated by a thick black line.

The arrows indicate the direction of action of the different muscles, their insertions at the cornea showing also the rotatory action of the muscles.

The arrows marked oculomotorius point to the resulta influence of the action of all the muscles supplied by the third nerve. The components are separated by the pinterolated drawing showing the diplopia from isolated paralysis cf the internal rectus.

The figures in black represent the true images, those with cross-lines the so-called false images of the affected eye.

The dotted lines within the larger squares are lines of separation between single vision with both eyes and double vision.

To compare the statements of a patient (looking at a lighted candle with both eyes, a red glass being held before one), the examiner should be behind the patient if the left eye is affected, but in front of him if the right eye. On mapping out the double images as stated by the patient these double images will agree in either case with those as given by Roth.

nerve nucleus is connected with the sub-nucleus of
the third for the internal rectus of the other side.
The sixth nerve nucleus thus acts as the centre
for the conjoined action of both eyes in lateral
movements of the eyes, and the connection be-
tween the two nuclei is held to be *via* the pos-
terior longitudinal bundle or fasciculus, a bundle of
nerve fibres passing down from the region of the
third nerve nucleus to the lower part of the medulla.

Conjugate deviation of the eyes to one side is a
symptom of high value if correctly interpreted. It
may be due to a cortical or sub-cortical lesion, or
to a pontine one; in either case it may be para-
lytic or irritative. A paralyzing lesion in the cere-
brum may cause besides hemiplegia paralysis of the
opposite sixth nerve with associated inaction of the
internal rectus on the side of lesion. Hence there
then will be conjugate deviation of the eyes to the
side of the lesion. If the lesion be an irritative one
the opposite sixth nerve nucleus overacts, and with it
the internal rectus of the side of the lesion. There
is then conjugate deviation from the side of the le-
sion. "The patient looks towards the convulsed mem-
bers if there is irritation; he looks towards the le-
sion if it is a paralyzing one." (Landouzy, Grasset.)

The rule is reversed in the case of a pons lesion
causing conjugate deviation of the eyes.

The Pupil. The pupil is the circular aperture in
the iris. The iris is a muscular curtain separating the
anterior from the posterior eye-chamber. It is com-

posed of circular fibres termed collectively the sphincter pupillæ, which are innervated by a branch of the third nerve, and radiating fibres forming the dilator pupillæ under control of the sympathetic.

The two sets of fibres are antagonistic and their combined action is to regulate the size of the pupil and hence to regulate the amount of light entering the eye.

When an increase of light falls upon the retina, besides the sensation transmitted to the occipital cortex an impulse is also sent through the optic nerve to the anterior pair of the corpora quadrigemina, each corpus receiving such impulse from both eyes. From the cells of the anterior corpus a new impulse is sent to the anterior part of the third nerve nucleus below, from which in turn a motor impulse is sent to the sphincter pupillæ, with consequent contraction of the circular fibres, and hence diminution of the size of the pupil. Lessening of the amount of light entering the eye acts negatively by lessening the demand on the third nerve nuclei, and then the tonus of the dilator fibres antagonizes the now passive sphincter, and the pupil dilates. The fibres or connections between the anterior corpora bigemina and the third nerve nuclei have not as yet been anatomically demonstrated. Since both anterior corpora receive the light-impulses for pupillary action from each eye, it can readily be understood why both pupils must normally be of the same size, even if light be admitted to one eye only.

When the visual axes are made to converge, as in

accommodating for near objects, the pupil contracts in associated action; when accommodation is relaxed the visual axes become more nearly parallel and the associated action of the sphincter pupillæ ceases, and the pupil dilates. Hence in testing pupillary activity under the stimulus of light, the subject must be made to look at a distant object, 10 feet away, if possible.

From the foregoing it is plain that alteration in the actions of the pupil, apart from adhesions of the iris, etc., must be due either to an affection of the third nerve or its nucleus or its cortical area, or a lesion involving the connection between the anterior corpus and the third nerve nucleus, or else to the sympathetic.

Irritation of the sympathetic fibres for the iris causes dilatation of the pupil; paralysis of these fibres causes contraction through the now unopposed action of the sphincter. Test for the unimpaired action of the sympathetic supply to the iris is made by stroking sharply or pricking or faradizing the skin at the side of the neck; dilatation of the pupil of the same side then occurs if the sympathetic supply is unaffected. The reflex is known as the pupillary skin reflex.

In facial neuralgia, in migraine, in some forms of headache, with chronic disorders of the teeth, etc., the pupil of the affected side may be altered as to size. Even affections in distant parts (mediastinal tumor) may so affect the sympathetic nerves as to alter the size of the pupil of the same side.

When the radiating fibres are contracted (irritation of the sympathetic) to a high degree, the pupil is

dilated, but some contraction is possible under the influence of light and in accommodation.

When the radiating fibres are paralyzed (paralysis of the sympathetic) the pupil is moderately contracted, but can contract still further under the influence of light and accommodation.

When the sphincter is paralyzed (third nerve) the pupil is dilated, but contraction under the influence of light and accommodation does not occur. The skin reflex is, of course, not affected, and the action of atropine, which besides paralyzing the sphincter also irritates the radiating fibres, increases the dilatation *ad maximum*. Duboisine, hyoscyamine and many other mydriatics act in the same way.

Spasm of the sphincter causes marked narrowing of the pupil, which is immobile under the influence of light and accommodation or even the skin reflex, the sphincter fibres being more powerful than the dilator ones. Eserine increases in such cases the myosis by irritating the sphincter and paralyzing the radiating fibres. Pilocarpine, muscarine and nicotine act similarly.

When only one set of fibres is paralyzed, the unopposed action of the other set results in its contraction, which increases by continuance, so that in course of time the dilatation from third nerve paralysis, for instance, becomes greater than at first.

Paralysis of both sphincter and dilator fibres causes moderate dilatation, with immobility of the pupil (cadaveric position).

The reaction of the pupil under the influence of light upon the retina may be lost, while the associated action in accommodation and convergence is retained. This is known as the Argyll-Robertson pupil, and is found in disease affecting the central gray below the aqueduct of Sylvius, the sub-nucleus for accommodation and convergence escaping. The condition is often present in locomotor ataxia.

Spinal myosis is the term given to loss of the sympathetic influence in the same disease; when present, the pupil is contracted often to the size of a pin-head, and may or may not have the character of the Argyll-Robertson pupil.

It must be remembered that in elderly persons the pupil becomes small, and its reactions slow or nearly disappear; the skin reflex shows the loss first. Dilated pupils in the old are pathological, whether from local ocular trouble or from nervous disease. On the other hand, contracted pupils in persons under fifty must be interpreted in a similar way.

The Ophthalmoplegias. The word ophthalmoplegia is used to indicate paralysis of the eye muscles. When only the external muscles of the eye are affected it is called ophthalmoplegia externa, when only the muscles within the eye are involved it is o. interna; o. partialis and o. totalis indicate respectively paralysis of some of the muscles and of all.

The source of paralysis of an eye muscle may be a lesion of the related nerve within the orbit or in

its course after emerging from the brain, or between the latter point and its nuclear origin, or finally in the nucleus itself. In any case the affection may be acute or chronic.

When a muscle whose function is to turn the eyeball is paralyzed, the eyeball can no longer be directed as formerly by the normal activity of the muscle, and the eye lags behind its fellow when the attempt is made to view an object lying in such direction. As a result the rays of light from the object do not fall upon the yellow spot of the affected eye, as in the normal eye, and two images are seen, because for the production of one image in binocular vision the visual axes must be so directed that rays from the object fall upon the same parts of the two retinas.

If, for instance, the affected eye be unable to turn outward, then in any effort to look in that direction the eyes are crossed, but the image seen by the affected eye is apparently still further in the direction in which the vision has been directed. For, as the rays of light cross in entering the eye it is plain that objects situated higher than the pupil must affect the lower segments of the retina. The brain has learned by experience to associate impulses received by the lower portion of the retina with objects situated above the eye; similarly, impulses received from the upper portions of the retina are recognized as being made by rays from objects below the pupil, those from the left half of the retina and those from the right half representing objects in the opposite half of the visual field in each case.

The importance of correctly determining the position of the image seen by the affected eye both absolutely and in relation to that seen by the unaffected one, is so great in the examination of certain forms of nervous disease that study of the figure reproduced from A. Roth is urged upon the reader.

When only one of the ocular motor nerves is paralyzed, the inability of the affected eye to follow the examiner's finger to the normal limit in some one direction will permit of a ready diagnosis. When two nerves are affected the same method will apply; when all three nerves are paralyzed the immobility of the eyeball will make the fact plain; a paretic state of the muscle or muscles may not show when tested as above directed, but yet may give double images under the test with candle and colored glass.

The causes of ocular palsies are chiefly rheumatic influences (cold) and syphilis directly affecting the nerve or indirectly causing pressure from gummy meningitis at the base. Diphtheria not infrequently is followed by paralysis of one or more eye muscles, usually the iris and ciliary muscle, more rarely the internal recti as well. In locomotor ataxia, abnormalities in the action of the muscles of the eye are quite frequently seen; in the early stages these paralyses are usually temporary.

Complete paralysis of the third nerve results in loss of power in the internal, superior and inferior recti, inferior oblique, the ciliary muscle, the circular fibres

of the iris and the levator palpebræ. When the fallen upper lid is raised, it is seen that the eyeball cannot move except outwardly and also downward and outward. The pupil is dilated but not *ad maximum;* it does not react reflexly to light nor in the effort at accommodation, the latter faculty being of course absent. The eyes are divergent, but the diplopia is crossed. Only a part of the third nerve supply may be affected; hence at times a single muscle only is paralyzed. A slight ptosis may be caused by paralysis of the sympathetic, the fibres of Müller's muscle thereby becoming paralyzed; at the same time the dilator fibres of the iris being paralyzed, the unopposed action of the sphincter causes contracted pupil.

Hysterical ptosis occurs, usually with associated spasm of the orbicularis; and a functional ptosis noticed on waking in the morning is sometimes observed, which soon passes off.

Paralysis of the fourth nerve is not often seen. The resulting diplopia is in the lower and outer half of the field and is especially annoying in walking, descending stairs or when at work. Downward movement of the eye can be made, but then the eye turns somewhat inwards so that the visual axes cross and the diplopia is therefore homonymous.

Paralysis of the sixth nerve occurs next in frequency to that of the third. The visual axes are crossed and the diplopia is homonymous.

Nuclear Ophthalmoplegia. Under the title polioencephalitis superior acuta (in contrast to p. inferior,

one of the bulbar affections) Wernicke has described a disease, acute in onset and rapid in course. Beginning with headache, vertigo and vomiting, delirium or it may be somnolence, occurs, and at this time paralysis of the muscles of the eye appears; in some instances almost total ophthalmoplegia was finally manifest, the sphincter iridis and levator palpebræ being unaffected. Neuritis optica occurred in some cases and ataxic gait, similar to that of cerebellar disease, was noted. No increase of temperature was recorded, but the pulse was usually rapid.

Most of the cases have ended fatally within a week or two.

Post-mortem examination revealed an acute hæmorrhagic encephalitis of the central gray at the floor of the third ventricle and beneath the aqueduct of Sylvius.

The cause assigned in the early cases is chronic alcoholism; later, cases have occurred after infectious diseases, especially influenza, from poisoning by spoiled sausage, by carbon monoxide and by nicotine.

Progressive nuclear ophthalmoplegia as an independent affection is rare; as an accompaniment or part of some organic disease of the nervous system it is not infrequent, especially in bulbar paralysis, locomotor ataxia, dementia paralytica and progressive muscular atrophy; it has also been observed in multiple sclerosis and in combined system-disease of the spinal cord.

The disease is gradual in its onset, in most cases a slight ptosis with some diplopia being the earliest

symptoms. With the progress of the disorder, other muscles are involved gradually. The interior muscles of the eyes escape in most cases, so that the affection is generally an ophthalmoplegia exterior bilateralis.

The prognosis is unfavorable for cure; the disease process may extend over many years, but it may cease to advance. The danger to life becomes greater when the process seizes the motor nerve nuclei in the medulla, or when it is a part of progressive muscular atrophy.

Anatomically, the disease is a chronic inflammatory or degenerative process in and about the ocular nerve nuclei beneath the floor of the aqueduct of Sylvius, the anterior sub-groups for the innervation of the ciliary muscles and the sphincters of the irides apparently escaping in most of the cases. The syphilitic process can affect the same region; such cases may improve under treatment. In multiple sclerosis the nuclei are not affected, as a rule, but the nerve roots are the seat of the trouble (Oppenheim).

Treatment should be that of the associated disease, but as the latter is frequently initiated by the former, the treatment then must be symptomatic. At the New York Ophthalmic Hospital, *Agaricus, Gelsemium* and *Physostigma* are favorite remedies in paralysis of eyeball muscles. *Causticum* and *Conium* are especially indicated in isolated ptosis and in paralysis of the ciliary muscle.

When the affection is even probably nuclear, *Syph-*

ilinum high, *Plumbum* and *Carboneum sulphuratum*
should be studied, while if the trouble be part of
a more extensive degenerative process, the remedies
applicable in the latter may find place here. Cases
undoubtedly syphilitic will require *Merc. jod. flav.*,
low, and if gumma be suspected it must be removed
by the solvent action of iodide of potassium in ma-
terial doses.

Other remedies that have proven valuable in treat-
ing paralytic eye muscles are *Rhus tox.* and *Arnica*,
prescribed on rheumatic or traumatic indications,
Jaborandi, *Natrum mur.*, *Nux vomica*, *Senega* and *Sul-*
phur. *Euphrasia* and *Phosphorus* have cured when
indicated by accompanying special symptoms or condi-
tions. In all cases the remedy should be sought for
by studying the known indications of the different
drugs.

Recurrent or **periodical oculo-motor paralysis** is
a somewhat rare affection with some peculiar features
that led Charcot to give it the name ophthalmo-
plegic migraine. It usually begins in youth or early
childhood, in one case at the age of 11 months. The
attack may be divided into two parts. First, severe
pain in, around and behind the eye of one side with
headache of the same side and vomiting. These
symptoms continue for a day or two, a week, or even
three or four weeks (Möbius), and they cease with
the occurrence of the paralysis, which may affect all
the muscles supplied by the third nerve. Occasion-
ally the external rectus has been affected. The pa-

ralysis lasts perhaps a few days, perhaps many months, but seems to be in direct relation to the length of the sensory part of the attack. The attacks return at definite intervals, in some cases a week, in others a month, in others six months, in some patients always at a menstrual period, and so on. In those having frequently recurring attacks, these are shorter. In the intervening time most of the paralytic symptoms slowly disappear, but partial ptosis or weakness of the pupillary reflex may remain after all other symptoms have gone. The intervals become less as years go on and the paralytic symptoms more permanent. The disease seems to be related to migraine, yet has positive differences from it. Migraine is at times accompanied by ocular palsies, but these are seen in the beginning of the attack, not after cessation of pain. With migraine there is in most cases inheritance, in the present disease none. Yet the present trouble is migrainous in its sensory manifestations. No satisfactory hypothesis of its origin has as yet appeared.

The treatment ought to be, in the first part of the attack, that for migraine; in the second part, that for paralysis of the third nerve. But the best results will be obtained by placing the patient under treatment during the interval and using, as they may be indicated, our great antipsorics.

Insufficiencies and other abnormalities of the ocular muscles have been assigned as causes of certain nervous affections, such as epilepsy, migraine, headache, chorea, facial and other spasm, vertigo and even organic nervous disease. Discussion of the subject has been extensive during more than a decade and repetition of it is out of place in this book. That an inborn insufficiency of some muscle of the eye can, in course of time, set up in a neurotic person many symptoms of nervous trouble may not now be doubted, but that *post hoc* means *propter hoc* in all cases in which graduated or other tenotomies were followed by relief, is not the writer's opinion.

Uncorrected errors of refraction may undoubtedly give rise to headache and other nervous trouble.

In either case of eye strain the concomitant nervous disorder may not be amenable to remedies until the ocular defect is removed or corrected.

Special treatment for the ocular conditions just mentioned belong properly to the ophthalmologist.

The fifth cranial nerve or **trigeminus** apparently emerges by two roots from the side of the pons. Its origin is analogous to that of a spinal nerve for the posterior or sensory root originates in the Gasserian ganglion, the cells of which give rise to the peripheral fibres and to the post ganglionic, afferent or sensory root. The fibres of the latter enter the pons and turn downward, ending each in a brush-work around the cells of the gelatinous substance in the medulla and upper cervical cord. In cross sections of the medulla

and upper cervical cord the fibres can be seen as a compact mass, a mere fringe outside the substantia gelatinosa at the level of the decussation of the pyramids, but as a large kidney-shaped bundle in the pons. Until recently it was termed the ascending root of the fifth.

The motor root of the fifth cranial nerve is divisible into a larger, the motor root proper, and a smaller root considered by Kölliker to be also motor, by others to be trophic in function. The latter arises from cells not aggregated into a nucleus, and placed at the outer limit of the gray matter around the aqueduct. The fibres from them can be seen in all sections as far down as their place of exit from the pons. They are called the descending root of the fifth. The distribution of the fibres is not known.

The motor root proper arises from the motor nucleus situated within the pons. Its fibres pass forward beneath the sensory fibres and their ganglion and are distributed to the masseter, the two pterygoids, mylohyoid, anterior belly of the digastric, the temporal muscles and tensor tympani. The motor root receives some fibres from the nucleus of the opposite side.

The trigeminus is the sensory nerve of the face as far back as the ear and as low as the lower outline of the lower jaw, of the scalp as far back as the crown, of the mouth and its contents, including the teeth, soft palate and uvula (but not the extreme base of the tongue); the antrum and the frontal sinus, part of the external auditory canal and the greater part of the

membrana tympani. The dura mater, which is the only sensitive membrane, is supplied by this nerve, except in the occipital region and the posterior fossa, and the tentorium and the falx are innervated by it.

The four ganglia situated on different divisions of the nerve are of some importance in connection with the sympathetic fibres which it receives; they subserve trophic, vasomotor and secretory functions.

The cortical area is unknown, but its sensory representation is probably in the parietal cortex, its motor representation being probably in the lower portion of the central convolutions of the opposite hemisphere.

Neuralgia of the fifth cranial nerve has been already considered under Neuralgia.

Trophic changes, such as herpes, in the distribution of the nerve are seen at times during an attack of trigeminal neuralgia. Herpes may be extensive and may affect the mucous membrane lining the mouth and give rise to additional pain in eating. Its cause is considered to be irritation of the Gasserian ganglion.

Paralysis of the nerve may occur from injury or from pressure by a tumor at the base. The result is anæsthesia throughout the distribution of the nerve, with paralysis of the muscles supplied by it. Chewing is done by the muscles of the opposite side, whose pterygoids carry the jaw toward the affected side. If irritation of the Gasserian ganglion be present, herpes of the cornea, followed by keratitis neuroparalytica (so-called) may be a result.

Treatment of such affections of the fifth nerve must be that of the underlying cause.

Progressive Facial Hemiatrophy.

A rather rare affection, characterized by wasting of the skin, subcutaneous tissues, bones, and at times the muscles of one side of the face. The affection develops usually between the tenth and twentieth years, and more frequently in girls. No cause is assignable in many cases, but in some, undoubted history of trauma at the affected site or a preceding infectious disease, can be obtained. The disease may show first at some one region, such as the neighborhood of the orbit. The skin becomes thinner and whiter (loss of pigment) or else becomes yellowish or brown (excess of pigment), the hair becomes finer, and later falls out. The subcutaneous connective tissue disappears and the skin becomes wrinkled. The disappearance of fatty and connective tissue from the bottom of the orbit permits the eyeball to sink and then the palpebral fissure becomes narrower—the eye appearing smaller than its fellow of the other side. The disease is slow in progress and when the bones are much involved, the asymmetry of the face becomes very striking. When the affection does not begin until adult age, the bones are but rarely attacked. The masseter muscles usually escape, but the muscles of the tongue are at times wasted. The electrical reactions in the affected muscles are not altered. The functions of the skin may be altered, the sebaceous secretion being lessened, while

secretion of sweat may be greater; the power of blushing may be lost on the affected area. Anæsthesia is not present as a rule, but pains in the distribution of the fifth nerve may precede the appearance of the atrophy. The disease progresses to a certain point and then ceases to advance. It has been seen as a complication of other nervous diseases and a similar atrophy in the arm may accompany it.

The pathology of the disease as ascertained by Mendel is, that it is due to a neuritis of the fifth nerve with degeneration of its so-called descending root.

Treatment of such cases as have apparently followed trauma, has some hope. In one case where the affection began soon after a blow beneath the eye, the trouble was arrested, arnica being given among other remedies, and galvanism used. The current was given stabile, the negative pole, an insulated carbon electrode, being placed upon the atrophied masseter muscle inside the cheek. The patient was under treatment several months and now, after the lapse of some years, the asymmetry of the face is hardly noticeable.

The **facial** or **seventh cranial nerve** is made up of the neuraxones of the cells of the facial nucleus in the pons. The fibres pass, while still within the pons, inward and upward, curving around the nucleus of the sixth nerve to pass downward and outward, finally emerging at the lower edge of the pons between the olivary and restiform bodies of the medulla. It enters a bony canal in the temporal bone, where it is joined by the chorda tympani

given off from the lingual branch of the fifth, and finally emerges at the stylo-mastoid foramen.

The facial nerve is the motor nerve for the muscles of the face, the platysma, the stapedius and the stylo-hyoid muscles.

The chorda tympani carries taste fibres,* which after joining the facial probably leave it by the great petrosal to once more become part of the fifth.

The facial area in the cortex is in the lower part of the motor area, and chiefly of the anterior central convolution. The fibres from its cells pass inwards to the internal capsule, and take the most inward position in the motor tract, which position they hold in the *pes* of the crus. At the beginning of the lower third of the pons they cross and end at the nucleus of the facial nerve. They are the first fibres of the motor tract to decussate, and so furnish means for an important difference in diagnosis between a cortical lesion and a lower pons lesion.

Peripheral Facial Paralysis.—Bell's Palsy.

The facial nerve being wholly motor in function (except that in part of its course it carries taste fibres from the lingual nerve which leave the trunk *via* the great petrosal nerve), disease or injury to the nerve is shown

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* There is much reason for holding that the sense of taste is carried by the fifth cranial nerve (lingual branch) chorda tympani, great petrosal and the second division of the fifth (but individual differences exist) from the anterior two-thirds of the tongue, and that the posterior third is subserved by the ninth or glossopharyngeal. Both nerves are nerves of common sensation also.

by paralytic symptoms in the muscles of the face. Such paralysis differs from paralysis of the face of cerebral origin; in the latter case the upper part of the facial distribution is usually not affected, the nerve supply through the trunk of the facial not arising from the cells of its nucleus, but from some higher source, and not forming part of the motor tract. Hence in a hemiplegia the orbicularis oculi, the temporal and the corrugator muscles are not paralyzed.

Peripheral facial paralysis most commonly results from exposure to cold and especially to the local influence of a draught of air upon the face. It also occurs in ear disease, from injuries to the region of the ear and from accidental section of the nerve in operation on the parotid gland. It has been recorded as part of multiple neuritis and in locomotor ataxia and at times in the infant from pressure of the forceps during delivery.

The symptoms appear within a few hours or a day or two after exposure, preceded at times by pain in the region of the ear (local implication of the fifth nerve). The most marked objective symptom is the inability to close the eye of the affected side; even the ordinary winking reflex is gone. The forehead cannot be wrinkled, nor the eyebrow brought to the middle line, as in frowning. The angle of the mouth on the affected side is lower than its fellow of the opposite side; in blowing, the air escapes from this angle of the mouth instead of the middle. In chewing, the food cannot be properly moved and accumu-

lates between the teeth and the cheek from loss of
power in the muscles of the latter. The dilator
muscle of the nostril of the affected side being para-
lyzed, that nostril is narrower than its fellow. The
lower lid not being in close apposition to the eyeball,
the tears do not get into the lachrymal canal and may
overrun the cheek, giving rise to redness and irritation;
the eyeball being always uncovered (even during
sleep) it is not kept free from minute particles, which,
together with evaporation, tend to set up irritation
or even inflammation of the conjunctiva.

The course of the nerve, its association for a short
distance with the eighth nerve, its own branches (es-
pecially the chorda tympani) permit of fine topo-
graphical diagnosis as to the site of the lesion, which
is valuable for the prognosis. Between the junction
of the chorda tympani below and the geniculate gang-
lion above, run the taste fibres. A lesion in the lower
part of the bony canal or after emergence of the nerve
from it, is not accompanied by loss of taste. A lesion
higher up in the canal may have temporary loss of
taste with lessening of the salivary secretion and hence
dryness of the mouth.

Rheumatic forms of facial paralysis are generally
peripheral and relatively slight in degree. The elec-
trical reactions of both nerve and muscles are not
changed, and rapid cure (within a few weeks) is to
be expected. When the nerve at the end of two
weeks shows a lessening but not complete loss of
reaction to galvanism, while the muscles give slow

contraction to direct application, without reversal of the formula, there is partial RD, and favorable prognosis of cure within three or four months may be given.

When complete RD of nerve and muscles is shown by the electrical tests, cure may occur in a few months or perhaps not at all.

If the lesion is due to ear disease, disease of the bone, etc., cure cannot take place until such disease ceases. If the originating cause was section of the nerve (operation, or other injury) a secondary operation to suture the divided ends together will be necessary before restoration of function can be effected.

In severe cases, after some months, secondary contractures affect the paralyzed muscles, the mouth being drawn up on the paralyzed side, and, to a casual observer, the really unaffected side then appears to be the paralyzed one.

Anatomically, Bell's palsy is due to degenerative peripheral neuritis of the facial nerve; the symptoms may occur in bulbar disease (q. v.). Disease of the nucleus alone will not cause involvement of the muscles surrounding the eye.

Gummy exudations at the base may involve the emerging fibres of the facial, but the auditory nerve will then hardly escape, and other and distant cranial nerves may be affected. The writer has reported a case of paralysis of the 3d, 5th and 7th nerves in a syphilitic; here, while there was inability to close the eye completely, there was also partial ptosis.

The homœopathic treatment of Bell's palsy must

be guided largely by the causal indications. In the writer's experience *Rhus tox.* and *Dulcamara* have given apparently good results. *Causticum* in one case and *Cimicifuga* in another acted similarly. *Aconite, Bellis perennis, Ruta grav.* and *Belladonna* are remedies that ought to be thought of in the rheumatic form of the affection. Since many cases are light in degree and would probably get well of themselves, it will need many such recorded cases to enable us to say definitely just how much our remedies accomplish.

In the cases that are symptomatic of some other disease as the cause, treatment for the latter is, of course, of the first importance.

The employment of galvanism in facial paralysis is a routine matter with the writer. Patients seem to experience benefit from it.

To protect the eye the writer instructs patients to close down the upper lid with the hand many times in the day and to sleep with a well-applied bandage over the closed lid. Further, patients should rub the paralyzed side of the face upward often during the day.

In old persons, peripheral facial paralysis may be very intractable.

Spasm in the distribution of the facial nerve will be considered with the spasmodic disorders.

The eighth cranial nerve is the common trunk of two nerves, distinct in origin and in function. One arises in the ganglion cells of the ganglion spirale of the cochlea, from which fibres can be traced out-

ward to the cells of the organ of Corti and inward
to the medulla. This is known as the cochlear nerve
and is the real auditory nerve.

The other arises from cells in the intumescentia
ganglioformis Scarpæ or vestibular ganglion in the
superior fossa at the bottom of the internal auditory
meatus. Fibres go from the cells in two directions,
in one to the vestibule and semi-circular canals, in
the other to the medulla. This nerve is known as
the vestibular nerve, or space-sense nerve, since it
carries impulses from the semi-circular canals in
which any change of position of the head results in
alteration of the relation between the membranous
duplicate of the semi-circular bony canals and the
fluid within them. The two nerves while yet within
the internal auditory meatus unite into a common
trunk, which reaches the medulla and enters it by two
divisions, the lateral and posterior one being the cochlear,
the inner and anterior being the vestibular nerve.

The greater number of the fibres of the cochlear
nerve end amid the cells of the ventral or accessory
acoustic nucleus, while a portion wind around the
inferior peduncle of the cerebellum to reach the
chief nucleus and Deiter's nucleus.

The fibres of the vestibular root pass to the inner
side of the ventral nucleus and reach the chief nu-
cleus and Deiter's nucleus, amid the cells of which
most of them end.

From the ventral nucleus new fibres arise that
cross in the raphe, pass into the lateral lemniscus in

part, in part to the formatio reticularis, and end about the cells of the posterior corpus quadrigeminum and internal geniculate body, whence a new set of neurones begin, whose long fibres go to the cortex of the temporal lobe. A part of the fibres do not cross, but pass up on their own side.

Fibres from the chief nucleus and Deiter's nucleus are sent to the cerebellum to end at the roof nucleus, or the emboliform and globose nuclei (Flechsig); a part enter the formatio reticularis, or enter the raphe and get to the formatio reticularis of the other side; another part crossing, enter the lateral lemniscus. The cortical area in relation to the vestibular nerve and its central tracts is unknown, but is placed by Bechterew in the parietal lobe.

Irritation of the nervous structures in the cochlea, or of the cells in the temporal lobes, will give rise to dysacusis, a condition in which ordinary sounds are perceived painfully, as seen during meningitis, headache, etc., and at times in hysterical states. Irritation of the same structures may also cause subjective sensation of sounds, as `whistling, buzzing of insects, singing or ringing, etc. Such subjective sensation is termed tinnitus aurium. Its most frequent cause is affection of the middle ear, circulatory disturbance, etc. It may be constant, and often is the basis of auditory hallucinations in the insane.

Destructive changes in the auditory nerve or its end organs in the cochlea give rise to loss of hearing power, either total or partial. Affections of the

external and middle ear can give rise to deafness by
preventing or interfering with the conduction of sound
waves to the cochlea. Such deafness is to be dis-
tinguished from nervous deafness by examination with
the otoscope and head mirror, and by the use of
the tuning fork. When a tuning fork is set in vi-
bration and its handle applied to the mastoid process
the bone will conduct the sound-waves to the cochlea
under normal conditions. Under the same conditions
when such perception of sound ceases, if the still vi-
brating instrument be removed and held near the
external auditory meatus, the sound will be perceived
anew. Thus bone and aerial conductions are both
present, the latter being, of course, the better. If
bone conduction is the better, the defect is in the
middle or outer ear. If bone conduction is lessened
or lost, the trouble is in the nerve or its endings.

When the semi-circular canals or the fibres from
them are irritated, false sensations are received from
them by the cerebellum and cerebrum; the patient
feels as if turning around, or as if falling in one di-
rection or another. Such vertigo is known as aural
vertigo if symptoms of auditory character are at the
same time present.

Ménière's Disease,

so called, is an extreme form of aural vertigo,
and is characterized by sudden attacks of violent
vertigo, so violent at times that the patient feels
it as a blow behind the ear, and may be

thrown to the ground. At the same time there is a
loud and often shrill noise in one ear. The sufferer
becomes pale, the face covered with sweat, and at
the end of the attack, which may last in the begin-
ning but a few minutes, there is vomiting or nausea.
Consciousness is not lost, but in some cases mental
confusion, with darkness before the eyes, is present.

The symptoms are due to an affection of the
labyrinth, such as hæmorrhage (injury), inflammation,
syphilis, etc. The manifestation of the trouble in at-
tacks is difficult to understand as part of a pro-
gressive disease, and they have been ascribed to
variation of intra-labyrinthine pressure, due to changes
of vaso-motor influence.

In most of the cases deafness in some degree
and tinnitus had been present, but occasionally deaf-
ness did not appear for some hours or some days after
the first attack.

The attacks tend to increase in frequency, and
finally the patient may be confined to bed, since
the vertigo becomes continuous on movement. When
the deafness becomes absolute the vertigo ceases as
a rule, because the labyrinthine nerve organs have
then been destroyed.

That the symptoms can come from affection of the
eighth nerve trunk or its so-called roots, is proven
by F. Alt's report of a case in which the symptoms
came on, and after the attack hearing was nearly
lost. Total deafness existed two weeks later, not-
withstanding which the patient kept to his bed on

account of the recurring vertigo. He was found to
be suffering from leucocythæmia, and died in three
months.

The autopsy supported the diagnosis of leucocy-
thæmia. Microscopic examination showed the middle
ear to be intact, the labyrinth without evident
change, but the inner and outer nerve roots showed
spots of small-celled lucæmic infiltration and the
trunk of the nerve slight degeneration. Here then
was a case with well-marked Ménière's symptoms
in which the labyrinth was not involved. In another
way the case is worthy of note, and, perhaps,
makes a point of diagnostic import between affec-
tion of the labyrinth and that of the nerve—deaf-
ness, even when total, was not accompanied by ces-
sation of the vertigo.

Diagnosis. In severe cases of Ménière's syndrome,
epilepsy may be erroneously diagnosticated. The
sudden fall, the partial (even momentarily complete)
loss of consciousness would make us suspect the pres-
ence of the latter disorder, but deafness is in favor
of the former. Dr. v. Frankl-Hochwart maintains
that cases of epilepsy occur in which the attack
is ushered in with vertigo, tinnitus, nausea and vomit-
ing, but without loss of hearing. Cases of epi-
lepsy with Ménière's triad as the aura, cannot be
differentiated from the genuine Ménière's disease if
demonstrable ear affection is present.

Treatment of any form of aural vertigo must be
largely influenced by discoverable cause, such as

middle ear disease, affection of the Eustachian tube, syphilis, etc. Symptomatically, *Natrum salicylicum* and *Chininum sulphuricum* are both well indicated and have repeatedly benefited. A typical case, which had been in the hands of several aural specialists, was relieved entirely and rapidly by the writer's prescribing *Chenopodium anthelminticum cum Terebinthina* (in 3d dilution), the mixture having been that taken by the involuntary prover. The deafness was intense, the patient only hearing conversation if it was very loud, yet she could distinguish the tinkle of the door bell two stories below; this symptom is found in Allen's Encyclopedia, Vol. X., under *Chenopodium*.

Other remedies reported as serviceable are *Aurum metallicum*, *Ledum palustre*, *Tabacum* and *Theridion*.

The **glossopharyngeal** or **ninth cranial nerve** is a mixed nerve. Its sensory fibres arise from the cells of the petrosal ganglion in the foramen jugulare and pass outward to subserve with common sensation and the special sense of taste the posterior third of the tongue and with common sensation the root of the tongue, pharynx, soft palate, Eustachian tube, tympanum and upper part of larynx.

The centripetal fibres from the same cells pass inward and enter the medulla in the postero-lateral groove and reach the sensory nucleus where they end. The second neurone arises from the sensory nucleus and ends near the cells of the temporal lobe.

The motor fibres arise from the nucleus ambiguus, dorsal to the olivary body. They pass out in the

same groove with the sensory fibres and form a common trunk. They are finally distributed to the muscles of the palate, pharynx and the middle constrictor of the œsophagus.

Isolated disease of the ninth cranial nerve has not been observed, owing to the difficulty of detecting it. Irritation of its fibres would produce the globus sensation as seen in hysteria, but this is to be considered as cortical in origin. Paralysis of the nerve would cause loss of taste and tactile sensation in the posterior third of one-half of the tongue and loss of tactile sensibility in other parts named above. Its nucleus is affected in bulbar paralysis (q. v.).

The **tenth cranial** or **vagus nerve** is a mixed nerve having both motor and sensory fibres. The latter arise from cells of the ganglion jugulare (Henle), otherwise called the ganglion of the root (Quain, Gray) within the jugular foramen. They are distributed to the dura mater of the occipital region and posterior fossa, to part of the external auditory meatus, to the œsophagus, pharynx and stomach, to the larynx and whole respiratory tract.

The centrally directed fibres from the ganglion pass into the medulla below the glossopharyngeus and reach the sensory vagus nucleus below the floor of the fourth ventricle, practically continuous with that of the glossopharyngeus in front.

The motor fibres of vagus arise from the motor nucleus, the nucleus ambiguus (common to the ninth and tenth cranial nerves). The fibres pass out by

several rootlets, having been joined by some fibres
from the nucleus of the spinal accessory nerve just
back of the vagus nucleus, and are sent to the mus-
cular structures of the pharynx, larynx, œsophagus,
stomach and intestines, as well as to the trachea and
bronchi.

The vagus contains both accelerating and inhibit-
ory fibres for the respiratory centre, for the heart,
and for the vaso-motor centre.

Secretory fibres are sent to the abdominal viscera
and to the respiratory tract.

The wide distribution of the vagus fibres, the
many functions which they subserve, especially in
the thoracic and abdominal viscera, make affections
of this nerve, for the most part, subjects for the
consideration of the general practitioner.

Affections of the nerve will be described in con-
nection with the eleventh nerve.

The **spinal accessory** or **eleventh cranial nerve**
arises in part from the posterior portion of the motor
vagus nucleus in the medulla, from which, indeed,
as has been stated, fibres join the root of the vagus
while yet within the medulla. Some fibres from the
nucleus join the vagus after emergence.

Another portion of the nerve, the spinal portion,
arises from the cells of the lateral and of the outer
part of the anterior gray horn of the cord, their lowest
origin being about the level of the fifth cervical nerve.
The fibres pass outward through the lateral columns
by several roots and ascend, successive additions being

made at each higher level. The trunk thus formed passes into the cranium through the foramen magnum and then turns outward to emerge from the cranium by the jugular foramen. Within the skull the fibres of spinal origin are joined by those arising from the nucleus in the medulla, but upon the emergence of the common trunk the latter divides into two branches, an inner and an outer. The former joins the vagus, contains the fibres from the nucleus in the medulla, and innervates the muscles of phonation; the latter contains the fibres of spinal origin and innervates the sterno-mastoid muscle and the upper part of the trapezius.

Paralysis of the vagus includes the fibres of the spinal accessory from the medulla. In such paralysis the palate hangs down on the side affected, it is not raised in intoning, the speech becomes nasal and in swallowing liquids a part may escape by the nose. The pharyngeal muscles being affected, swallowing is difficult. The laryngeal muscles show paresis of the abductor of the vocal cord of the affected side, and if paralysis, the cord is in the cadaveric position and immobile. The heart's action may be accelerated, but this is usual only when the nerves of both sides are affected; the breathing then becomes slow and irregular.

Defective contraction of the stomach with faulty digestion and loss of the sensation of hunger can result from a paralyzing lesion of the nerve; irritating lesion has caused insatiable appetite.

Lesions of the nerve may be from affections of the base of the skull or of the upper vertebræ, from meningitis, syphilis, etc. Lesion of a division of the nerve can occur from injury, pressure of a tumor or by direct poisoning, as in diphtheria.

Spasm in the distribution of the vago-accessorius nerve may affect the pharynx and œsophagus (globus) or the larynx (laryngismus stridulus, larynx "crises" in locomotor ataxia).

Treatment of affections of the nerve must be purely symptomatic unless the cause can be traced (meningitis, syphilis, etc.). Reference to the section on Bulbar Paralysis may give some hints.

A lesion of the spinal or accessory portion of the eleventh cranial nerve causes paralysis of the sterno-cleido and trapezius muscles. In consequence the head cannot be turned to the opposite side, nor the shoulder of the affected side raised. The full contour of neck and shoulder region is changed and appears sunken.

Only the upper portion of the trapezius is completely paralyzed, the other portions being also innervated by the cervical nerves. The scapula is more distant from the spinal column than normal and is also rotated, the lower angle being nearer the spine than normal. Atrophy is present.

If both nerves are affected both sets of muscles that support the head are affected, and there will be inability to hold the head up, so that the latter falls

forwards or backwards in accordance with the pull
of gravity.

Lesion of the nerve within the skull or in the for-
amen magnum may be by pressure from exudation,
meningitis, etc. Below the foramen, disease of the upper
vertebræ and injury, etc., can affect it. In progressive
muscular atrophy its nuclear origin may become in-
volved.

Spasm in the distribution of the spinal part of the
spinal accessory nerve will be considered in the section
on Spasmodic Diseases.

Treatment of paralysis of this part of the nerve
must be first for any underlying cause or any in-
cluding disease (bulbar paralysis, progressive muscular
atrophy).

The **hypoglossal** or **twelfth cranial nerve** arises
from the cells of a nucleus beginning in the lower
portion of the medulla and placed antero-laterally to
the central canal. Towards the rest of the brain the
canal is gradually displaced dorsally until it merges
into the widening posterior fissure and opens out as
the fourth ventricle. The nucleus of the twelfth nerve
is placed obliquely, so that it gets nearer to the canal
and the opening of the fourth ventricle and its upper
part is below the floor of the latter and near the
median line.

From the long nucleus fibres arise which pass down-
ward and outward between the pyramid and the olive
to emerge as the twelfth nerve roots.

The nerve supplies the muscles of the tongue, both intrinsic and extrinsic and the genio-hyoid muscle.

Paralysis of the nerve after its emergence from the medulla may be by compression from exudation, tumor; in the foramen and lower down by similar conditions or by disease of the bone.

Within the medulla it may be affected (and usually bilaterally) in bulbar paralysis.

In paralysis of one hypoglossal nerve the affected side of the tongue lies higher at the base than does the other half; when protruded it deviates curved towards the paralyzed side. When moved within the mouth its back part deviates towards the unaffected side.

If both nerves are affected the tongue lies motionless on the floor of the mouth; speech and eating are greatly interfered with. For further description and treatment *vide* section on Bulbar Paralysis.

Fig. 27. The positions of the different nuclei of origin of the cranial nerves will be understood by study of the accompanying figure, which represents the structures between the anterior pair of the corpora quadrigemina and the upper portion of the spinal cord. The structures of the right half are seen. To the left and below *Rad. anter.* = anterior nerve root. *XI* and bracket are at the nucleus of the eleventh nerve; fibres pass dorsally and are seen at the side as rootlets. Near them is a line, finely dotted, the common ascending root of the eleventh and

Fig. 27.

tenth nerves. They pass under the end of the upper dark mass, which is the nucleus of the tenth nerve posteriorly and of the ninth anteriorly. Above this conjoint nucleus is seen the nucleus of the twelfth. In the middle of the picture, extending lengthwise, is seen a thick dotted mass traceable into the pons, where it turns down and out. It is the so-called ascending root of the fifth nerve. *Vs.* = sensory root of fifth nerve; *Vm.* = motor root, whose fibres can be seen coming from its nucleus, the dark egg-shaped mass above. From the sensory root of the fifth a thick line can be traced upward into the region beneath the corpora quadrigemina; it is the descending root of the fifth. The long dark mass in the middle beneath the nucleus of the ninth and tenth is the large nucleus of the eighth nerve; its fibres are entering at *VIII*, passing under the other nucleus of the eighth nerve. Just above the latter nucleus is seen a scattered nucleus, that of the seventh nerve, whose fibres gather together and course over and around the sixth nerve nucleus. The roots of these two nerves are sufficiently well indicated. *IV*, near *corp. quad.*, shows both fourth nerves crossing. At *III* are seen fibres of the third nerve coming from its nucleus.

The Brain.

The Brain-Axis. By the term brain-axis is meant the brain structures between the lower end of the medulla and the optic thalami. It therefore embraces the medulla, pons Varolii, crura and the corpora quadrigemina with their included structures.

As many special nervous masses and fibre-tracts are mentioned in the present section and have been alluded to in the preceding one it is deemed advisable to consider here briefly the finer anatomy of the brain-axis and chiefly by the description of illustrations.

At the level of the first cervical nerve a section of the spinal cord shows some variation from the usual appearance of a lower section.
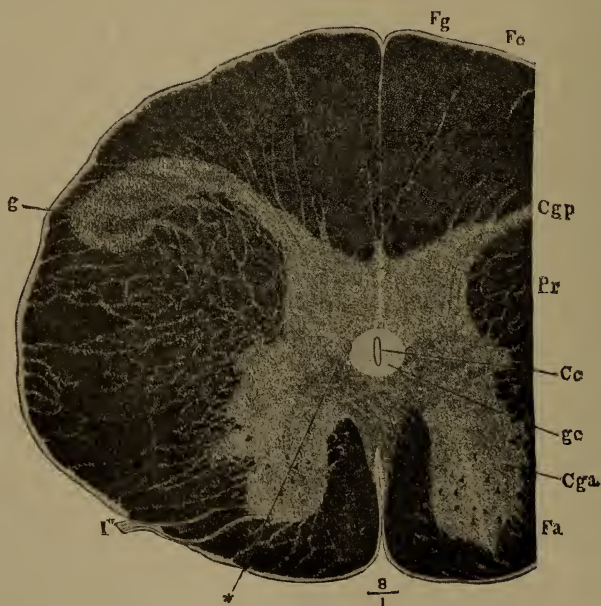
Fig. 28.

Fig. 28. From Henle. *Fg,* Fuuiculus gracilis or column of Goll ; *Fc,* funiculus cuneatus or column of Burdach ; *Cgp,* posterior cornu ; *Cga,* anterior cornu.

The ascending posterior columns have increased greatly, the posterior gray horns are thrust outward and have become smaller and at the end of each is seen, turned anteriorly, the large mass of gelatinous substance of Rolando.

A section in the lower part of the medulla shows the ending of the decussation of the pyramids, the fibres of which are seen crossing the middle to get into the lateral column of the opposite side. The anterior

Fig. 29.

Fig. 29. From Henle. Decussation of the pyramids. *Ng*, nucleus of Goll's column ; *g*, gelatinous substance at head of posterior gray horn.

gray horns are thus separated from the central gray matter. A spinal rootlet of the eleventh cranial nerve

is seen emerging at the left. In the column of Goll
the nucleus of that column is beginning.

Figure 30 shows a section of the medulla a little
higher. The decussation of the pyramids is just be-
ginning, a few fibres of each crossing. The nucleus
of the column of Goll fills nearly all the area formerly

Fig. 30.

Fig. 30. From Henle. Decussation of the pyramids at the level of the lower
roots of 12th cranial nerve. *Nc,* nucleus cuneatus or nucleus of column of
Burdach. Figs. 28, 29 and 30 show the gray matter in light tint, the white
matter in dark.

occupied by fibres and the nucleus cuneatus or nucleus
of the column of Burdach is quite large.
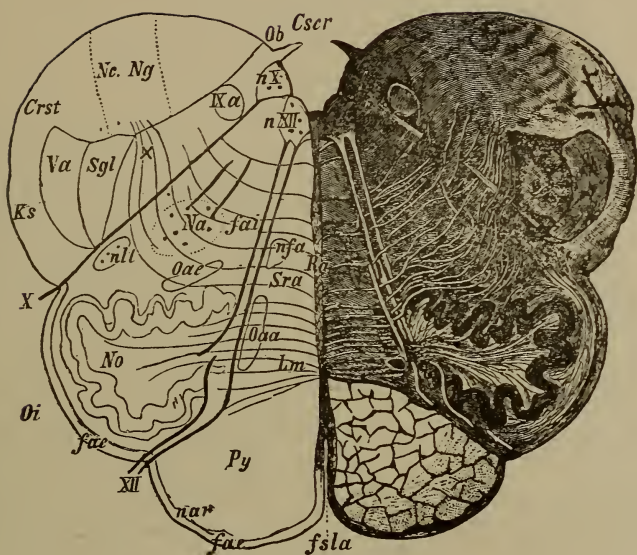
Fig. 31.

Fig. 31. From Obersteiner. The structures shown in the right half are explained diagramatically in the left half. *Na*, motor nucleus of ninth and tenth nerves. *IX a*, ascending root of ninth nerve. *Sgl*, substantia gelatinosa. *Va*, descending spinal root of fifth. *Crst*, the restiform body. *Ks*, direct cerebellar tract. *Oi*, inferior olive. *Lm*, lemniscus. *Py*, pyramid.

Figure 31 represents a section of the medulla through the calamus scriptorius. The opening above is the beginning of the fourth ventricle. The pyramids are intact. The olivary body, the tenth and eleventh nerves coming from their nuclei, the restiform body and the direct cerebellar tract are to be noted; fibres (at x) coming from the nuclei of the posterior columns are seen to curve to the middle line and cross. The twelfth nerves frame as it were between them the interolivary tract or lemniscus.
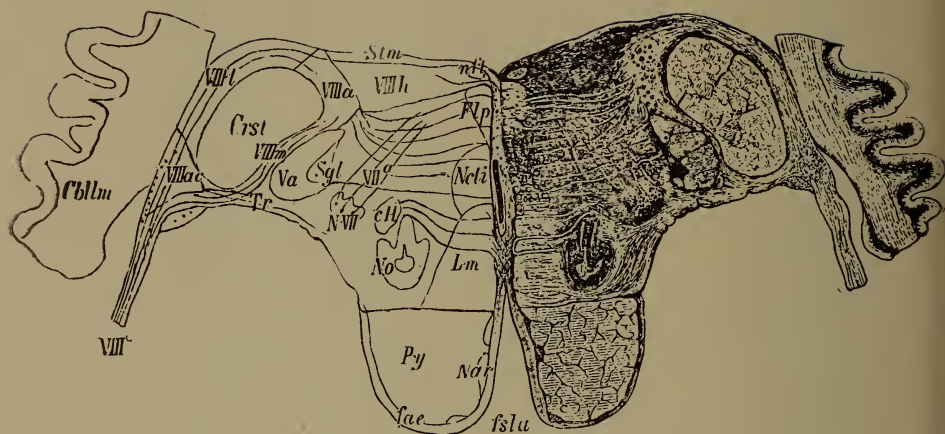
Fig. 32.

Fig. 32. From Obersteiner. *Cbllm*, part of cerebellum. *VIII*, the eighth cranial nerve. *VIII l*, lateral root. *VIIIm*, median root. *VIII ac*, accessory nucleus. *VIII h*, chief nucleus of the eighth. *N VII*, nucleus of the seventh. *VII a*, root fibres ascending from the same.

Figure 32 shows a section of the medulla made at the striæ acusticæ. The floor of the fourth ventricle is wide and flat, the covering cerebellum has been cut away except at the sides of the section.

The restiform body is seen surrounded by the roots of the eighth cranial nerve emerging as one trunk below. The seventh nerve nucleus is seen giving off fibres which here pass upwards.

Fig. 33.

Fig. 33. From Obersteiner (reduced). $N VI$, nucleus of sixth cranial nerve giving off fibres which descend (shown as thick lines). $VII b$, ascending fibres of seventh cut across. $VII c$, descending fibres of seventh. Ndt, nucleus dentatus of the cerebellum. Flp, posterior longitudinal fasciculus.

Figure 33 shows a section of the pons. The connections with the cerebellum have been cut away. The motor tract fibres are not in a compact mass as in the pyramid of the medulla, but are in bundles separated by the cross-fibres of the pons. A portion of the seventh nerve nucleus is seen at this level giving off some fibres which ascend. The fibres from the lower level have already ascended to turn around and over the sixth nerve nucleus, but the turn or knee is at a level a little higher and cannot be seen in this section, but as they turn backward they are again seen in the section, and pass downward. The restiform body has at this level become really the inferior peduncle of the cerebellum. The lemniscus is in a compact mass and in the field above is the formatio reticularis.

Fig. 34.

Fig. 34. From Obersteiner (reduced). *Lng*, Lingula ; *Vlma*, velum medullare anticum. *Brcj*, superior peduncle of the cerebellum. *Vd*,'descending root of fifth cranial nerve. *F'lp*, posterior longitudinal bundle. *Lm*, median lemniscus. *Lml*, lateral lemniscus. *Frtg*, formatio reticularis. *Py*, pyramidal tract separated into bundles by the cross fibres of the pons.

Figure 34 shows a section beyond cerebellar structures. The superior peduncle of the cerebellum is seen coming from its place of origin (?), to be shown in the next figure. The lemniscus has separated into two distinct bundles of fibres — the lateral or lower lemniscus, which goes to the region of the corpora quadrigemina, and the median or upper lemniscus, which terminates in the cortex of the parietal lobe. The formatio reticularis and the posterior longitudinal bundle are indicated.
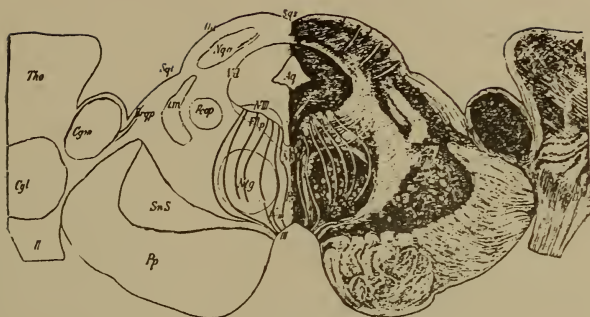
Fig. 35.

Fig. 35. From Obersteiner (greatly reduced). *Tho*, optic thalamus. *Cgm*, internal geniculate body. *Cgl*, external geniculate body. *II*, optic tract. *Pp*, pes or crusta of the crus cerebri. *SnS*, substantia nigra. *Ntg*, red nucleus of the tegmentum. *III*, fibres of the third cranial nerve. *N III*, nucleus of the third nerve. *Lm*, median lemniscus. *Aq*, aqueduct of Sylvius.

Figure 35 shows a section through the anterior pair of the corpora quadrigemina, the crura cerebri, through part of the optic thalami and of the external geniculate bodies. Around the aqueduct is seen the central gray at whose outer limit is the descending root of the 5th cranial nerve; below the aqueduct and lateral from it is the nucleus of origin of the 3d cranial nerve, from which the nerve fibres are going downward to emerge through the crus. They pass the posterior longitudinal bundle just beneath the central gray and through and around the red nucleus of the tegmentum. The lateral lemniscus has disappeared, the median one remains. The large mass of substantia nigra separates the pes or foot of the crus from the parts above, all the latter being included under the word tegmentum.
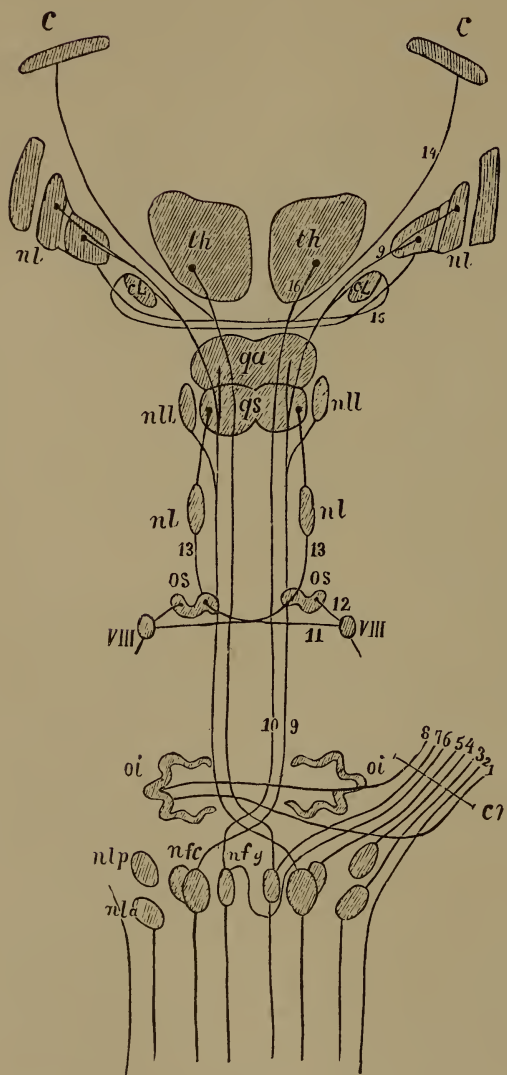
Fig. 36.   From Bechterew. A schematic representation of the course of fibres from the nuclei of the posterior columns of the spinal cord to and through both lemnisci and to the cerebellum.

C, the cortex ; nl, the lenticular nucleus ; th, the optic thalamus ; cL, Luys' body or corpus subthalamicum ; qa and qs, the corpora quadrigemina ; nll, nucleus in the lateral lemniscus ; os, the superior olive ; VIII, anterior acoustic nerve nucleus ; oi, the olivary body ; nla, nucleus of antero-lateral column ; nlp, nucleus of postero-lateral column ; nfc, nucleus cuneatus, at the top of the column of Burdach ; nfg, nucleus gracilis or nucleus of the column of Goll ; cr, restiform body becoming the inferior peduncle of the cerebellum. The fibres in the latter can be traced in the figure and need no explanation, except to say that fibre 2 represents the direct cerebellar column of the spinal cord.

The course of fibres from the nucleus of the lateral columns is not given. They get into the formatio reticularis (vide Figs. 31 and 34) and ultimately reach the parietal cortex.

Fig. 36.

Recent investigations by A. Hoche, of Strasburg, give new light on the course of the column of Gowers or antero-lateral tract.

His results support the conclusions of H. T. Patrick,

of Chicago, and others, who traced the fibres of this column in lower animals to the region of the corpora quadrigemina, where they turn backward with the superior peduncle of the cerebellum and end in its middle lobe. Hoche's investigations were on man, with the same result. Edinger had already proposed that the antero-lateral tract and the direct cerebellar tract be termed cerebello-spinal tracts, ventral and dorsal, respectively. Gowers' column, in view of these new facts, cannot now be considered as carrying pain- and temperature-sense impulses.
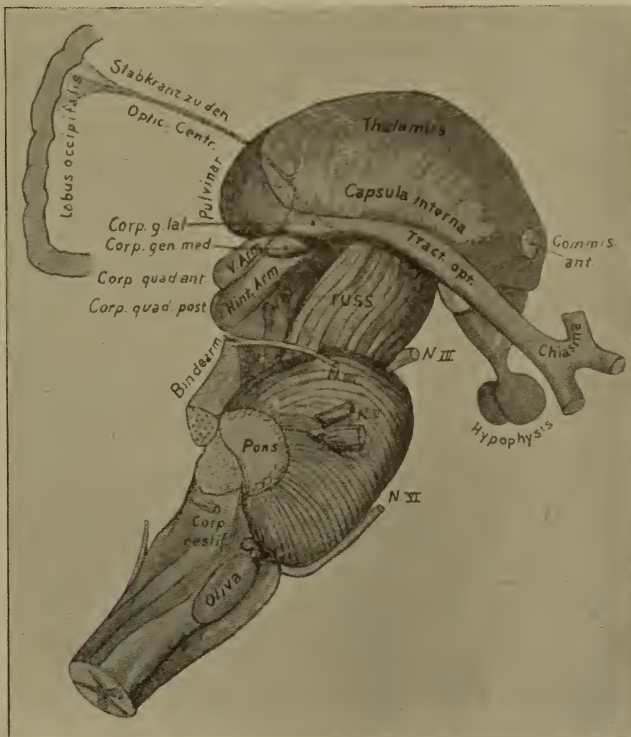
Fig. 37.
From Edinger.

Figure 37 gives a side view of the brain axis with the optic thalamus and part of the cerebral cortex. Fibres going to the occipital lobe from the primary optic centre are marked "stabkranz" (corona radiata). The left optic tract has been cut across, so that the chiasm with the remaining portions of the optic nerves is hanging to the right, out of its proper place. The word "fuss" is upon the foot or pes of the right crus cerebri. The geniculate bodies are indicated, the external or lateral one and the internal or median one being so designated. Of the corpora quadrigemina the positions of the two on the right side are shown and designated. The arm or brachium from each is seen. The three peduncles of the cerebellum are seen cut across, and their relative positions shown. The word "bindearm" is upon the superior, the word "pons" is on the middle, and the term corpus restiforme is upon the inferior peduncle.
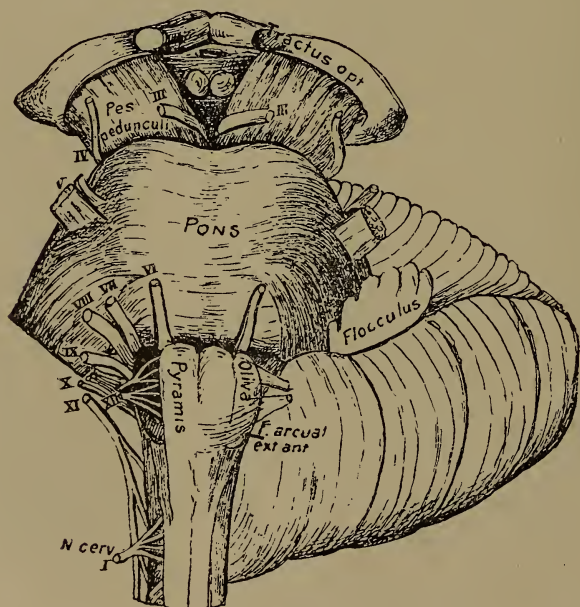
Fig. 38.
From Edinger.

Figure 38 gives a view of the inferior surface of the brain-axis with the left hemisphere of the cerebellum attached. At the optic tract in front, the chiasm and attached portions of the optic nerves have been turned backward. Pes pedunculi=pes or foot of the crus cerebri. The places of exit of the cranial nerves are marked by a Roman numeral in each case.

Progressive Bulbar Paralysis. — Glosso-labio-pharyngeal Paralysis. The disease is a progresssive degeneration affecting the motor nuclei existing beneath the floor of the fourth ventricle. Its cause is unknown; its relation to degeneration of the motor cells in the anterior gray horns of the cord has already been stated. It is seen mostly in the latter half of adult life, the majority of the cases occurring between the ages of 50 and 60. It has been ascribed to exposure to cold, to trauma, to over-use of the related muscles, to lead poisoning, syphilis, great emotional strain, and one case appeared after an attack of diphtheria.

Its onset is very gradual, the first symptoms being those of the tongue, whose finer movements in articulate speech become impaired, the sounds represented by the letters l, t, d, n and r being the first to become indistinct in enunciation. Later, the letters k, g hard and j are imperfectly uttered. The tongue may by this time show some gross changes; it cannot be freely or fully moved and may show some area of distinct atrophy. Later the lips become affected and the labials p, b, f, v, m, as well as the vowel o, cannot

be properly sounded. When the muscles of the soft palate become involved, a nasal tone is given to the speech, and from imperfect closure of the posterior nares in attempting to swallow liquids, the latter may come out of the nose.

The œsophageal muscles being affected, swallowing becomes a matter of difficulty, and, as there is imperfect closure of the larynx, there is danger of invasion of the larynx and bronchi by foreign matters and the consequent setting up of pneumonia. When the laryngeal muscles are well involved the voice is weak or veiled or even absent, while coughing is impossible or toneless.

The atrophy of the tongue increases so that its movement is eventually impossible, and as it lies at the bottom of the buccal cavity it usually exhibits a peculiar fibrillary tremor to be likened to a fine vermicular motion; furrows and depressions in the tongue show the atrophy. The lips also become atrophic, are incapable of closure, so that the mouth is always more or less open, and the patient cannot blow or whistle or smile. From the open mouth (the lower lip and chin hanging), the saliva drools constantly, while the muscles of the upper part of the face being unopposed, the naso-labial folds are more pronounced than normally and the face takes on an expression of pain. Sensation is not affected, except that a feeling of tension is felt in the muscles of the throat, but the pharyngeal reflex becomes lost.

The electrical reactions may show no change from

the normal until the disease has well advanced, and then only the partial reaction of degeneration.

The disease is slowly progressive, yet most of the cases end fatally within three years or so; cases have been reported in which the lethal termination occurred within the first year.

The fact that the degeneration is only part of a widespread process in the motor nuclei, whether of the anterior gray horns below or of the region of the aqueduct of Sylvius above, must be held in mind in considering the prognosis. The disease is incurable.

An **acute, apoplectic** form of **bulbar paralysis,** due chiefly to thrombosis or embolism, occasionally to hæmorrhage, exists. With this, since other parts in addition to the motor nerve nuclei of the bulbar region are involved, there are usually some additional symptoms as well as the suddenness or rapidity of onset, to serve as guides in diagnosis. Paralyses in the limbs, rapid pulse, disturbances of respiration and at times implication of the bladder, put the trouble outside the category of degenerative nuclear disease. Tumors affecting pons or medulla may by pressure give rise to many of the symptoms of bulbar paralysis, but the existence of syphilis, of cardiac affection, or of vascular disease, will direct the attention aright.

Pseudo-bulbar paralysis must be borne in mind as a possibility. It is due to symmetrically placed lesions in the cortex of both cerebral hemispheres or in the basal ganglia on both sides. The symmetry in location on the two sides will not be absolute and the apoplectic

onset, or the occurrence of stages of advance, will distinguish this form from the slowly progressing trouble due to degenerative disease in pons and medulla.

A special form of disease having bulbar paralytic (and sometimes atrophic) symptoms, mostly with some ophthalmoplegia, and in many of the reported cases muscular asthenia which may involve all the limbs, has been described by a number of observers. The disease may begin with ptosis (one-sided or bilateral) or some other muscular affection of the eye. Here, as also in all the muscles involved, the special feature is myasthenia, so that a few efforts tire out any muscle for the time being, and this exhaustibility appears under tests by the faradic current, the muscle responding fairly well at first, the response failing after a few contractions have been elicited. The condition worsens, but after a time, it may be months, improvement may set in and an apparent restoration occur. This recovery is believed to be deceptive and to mean only remission. Cases have died after return of the disorder following a complete apparent recovery lasting several years.

Post-mortem examination has so far revealed nothing to account for the symptoms.

The bulbar affection just described has lately attracted a great deal of attention. Oppenheim has termed it bulbar paralysis without post-mortem findings; Jolly, amyosthenia gravis pseudoparalytica; and Strümpell, asthenic bulbar paralysis. As Erb had re-

ported three cases of the affection in 1878, it has been called Erb's disease, but the name **asthenic bulbar paralysis** is generally accepted.

The treatment of bulbar paralysis must be largely guided by the diagnosis in any case. In the pseudo-bulbar paralysis (from cortical lesions) remedies must be addressed to such lesions if possible, and greater weight must be given to the non-bulbar symptoms.

In the apoplectic form, while the bulbar symptoms must be guides in choosing a remedy, nevertheless the character of the lesion, when this can be ascertained even as a probability, may make a change in the selection.

The true degenerative form will require the remedies that are known to produce similar degeneration. Dr. Berridge, of London, reported as a fact that workers in binoxide of manganese are frequently affected by bulbar paralysis. The writer has used this drug in one case without result, but in a case of the apoplectic variety, the symptoms having existed without material change for two years, the *Manganum binoxide* given three or four times a day in third trituration caused marked improvement in speech, power of swallowing, and other symptoms.

Lilienthal in his therapeutics (made up of clinical reports of cure or improvement) gives *Plumbum, Causticum, Cadmium sulph., Phosphorus* and *Nux vomica* as having been serviceable in bulbar paralysis, but without differentiating between types of the disease.

Two cases of the asthenic form are at present un-
der the writer's care. *Manganese binoxide* 3d trit.
was given in both cases without any perceptible ef-
fect. *Nux vomica* 2x seemed to hold the disease in
check, but one patient said it stimulated him, yet had
no effect on his speech or power of swallowing.
Hydrophobinum 200 (given because of the nerve cen-
tres affected) brought great relief, and then because
of the choking in a dangerous degree that came on
when laughing, *Stannum* 200 was prescribed, and with
marked improvement. Meanwhile, the speech has be-
come better, its tone less nasal, but some myasthenia
is present, so that he has to take a period of rest
before any business interview. It is typical of such
cases that their symptoms are always better in the
morning after a good rest, which brings *Phosphorus*
to mind as a remedy.

Prof. Jolly in reporting his cases, mentions the fact
that the symptom myasthenia as seen in them was
very like that produced by proto-veratrine. In a case
of bulbar affection not of the asthenic variety, but
due to syphilis and probably gummatous, seen by
the writer several times in consultation, the physician
had prescribed *Veratrum viride*, low, on the indica-
tions characteristic of that drug, especially those of
the tongue. Whenever he withdrew the remedy, the
bulbar and paralytic symptoms distinctly worsened.
The patient was supported for many weeks by rectal
feeding, and received large doses of *Potassium iodide*
in the same way, but notwithstanding the use of the lat-

ter drug, it is more than doubtful that his recovery from the dangerous bulbar symptoms, which resulted after a few months, would have occurred without the special influence of the *Veratrum*.

Diseases of the Central Nervous System.

Under the term central nervous system are included all nerve structures existing from the cerebral cortex down to, but not including, the ganglion cells in the anterior gray horns of the spinal cord and the places of entrance into the posterior part of the spinal cord by the posterior spinal nerve roots. The word brain means all the nerve structures within the skull cavity, and these are divisible into five pairs of organs, three of which are evident on inspection of the brain when removed from the skull; the remaining two, being buried within the brain mass, are recognizable after section as anatomical entities only by a knowledge of development.

The brain developes from five fundamental brain vesicles (originally three primary ones) in such way that the first (beginning at the nasal end) grows at such a rapid rate that it covers in all the others; the second and third develop to a less extent, retaining their original relatively central position, while the fourth developes laterally into two large masses; the fifth undergoes but little enlargement. The structures of the fourth and fifth, although covered in from above, are readily seen from below.

The first is the cerebral hemisphere; the second

the thalamus; the third the corpora quadrigemina anterior and posterior of one side, the fourth is pons and cerebellar hemisphere, while the fifth is a lateral half of the medulla. In each case there is a pair separated or nearly separated, one from the other, except in the third and fifth where the separation is only indicated by a more or less distinct raphe.

The functions of the brain structures are the aggregations of the functions of their individual neurones, and these functions are of two kinds — that is to say, the cell body either sends out or receives impulses. A centrifugal impulse or one from the cortex to the periphery, is a motor impulse; a centripetal one or one from the periphery to the cortex as the final or end station, is a sensory impulse.

Motor impulses are of one kind only—that is, they cause contraction of muscle fibre. Hence, it may be assumed that if a motor impulse from cells in a cortical area in relation to, say, the right leg, could be switched off, as it were, so as to reach a muscle of the left forearm, that muscle would be put into action. It is otherwise with sensory impulses. Here the end-organs are highly differentiated, so that qualities of heat, cold, simple touch, pain, the degree of flexion of a limb, or the amount of contraction of a muscle, can be in each case correctly appreciated. It is to be assumed that could the cells of the organ of Corti replace the rods and cones of the retina, the former would not be affected by the rays of light falling on the pigment layer—they were not

differentiated for such purpose. On the other hand, it may be that the end-organs of the skin are not so highly differentiated as has been just suggested, but that they receive different impressions and send inward impulses of a general character, which, in turn, are taken up by intercalated neurones acting as transformers, and passed on as fully differentiated qualities of sensation. In this way may be explained the existence of a chain of several neurones which exist in the pathway of sensory conduction; for the conduction of centrifugal motor impulses but two neurones are needed, both being long ones, the central neurone beginning as the cell of the motor area of the cortex and ending in the neighborhood of the large ganglion cell of the anterior gray horn of the cord, which cell is the beginning of the peripheral neurone, whose terminus is in the end plate upon a muscle fibre at the periphery.

The neural pathways between the cortex and periphery, both sensory and motor, form the projection system of fibres.

The optic thalamus is the fully developed second fundamental brain vesicle. It is in connection apparently with all parts of the cerebral cortex of its own hemisphere and sends also fibres downward, some of which join those of the motor tract whose cells act independently of volition. The thalamus is divisible into five large irregular and unequal masses, the larger posterior one being the pulvinar.

The third fundamental brain vesicle in developing

gives rise to the corpora quadrigemina and the struc-
tures below them, including the nuclei of origin of
the third and fourth cranial nerves. The vesicle
develops inwardly instead of outwardly, so that in-
stead of large ventricular spaces there is a channel of
small lumen known as the aqueduct of Sylvius, and
which is all that remains there of the original
neural tube. Below are the crura cerebri, not sepa-
rable anatomically from tissue above ; yet as they
are composed largely of the motor and sensory tracts,
together with other fibre tracts, they cannot be considered
genetically as derived from the vesicle in question.

The fourth vesicle develops laterally into two
masses, the cerebellar hemispheres, from each of
which fibres pass inwards, decussate in the middle
line, and go forward to the frontal lobe of the oppo-
site cerebral hemisphere.

The mass of fibres on each side and at the cross-
ing, form a considerable part of the bulk of the pons
Varolii, which also holds the motor and sensory tracts
in their passage downward and upward, respectively.
Still further increasing the bulk of the pons, are the
nuclei of the fifth and sixth cranial nerves.

The fifth brain vesicle develops into the medulla
oblongata, which contains anteriorly the motor tracts
now united into compact masses of fibres, known as
the pyramids of the medulla, the continuation upwards
of sensory pathways, the nuclei of cranial nerves from
the seventh to the twelfth, and specialized structures
known as the olivary bodies.

The **Cerebral Cortex** and the **Cortical Areas**. The cerebral cortex is composed so largely of cells that on section it appears gray. It is in folds or convolutions (also termed gyri); the depths or depressions between them are termed fissures (or sulci). The fissures appearing earlier in fœtal life are known as primary fissures and are an inch or more in depth. These are the fissure (so-called) of Sylvius, the fissure of Rolando, the interparietal—all on the outer face of the cerebral hemisphere, and on its inner face the parieto-occipital and the calcarine fissures. Between the cerebral hemispheres at the top is the great longitudinal fissure completely separating the two hemispheres in the frontal and occipital regions, but in the middle only as far below as the corpus callosum.
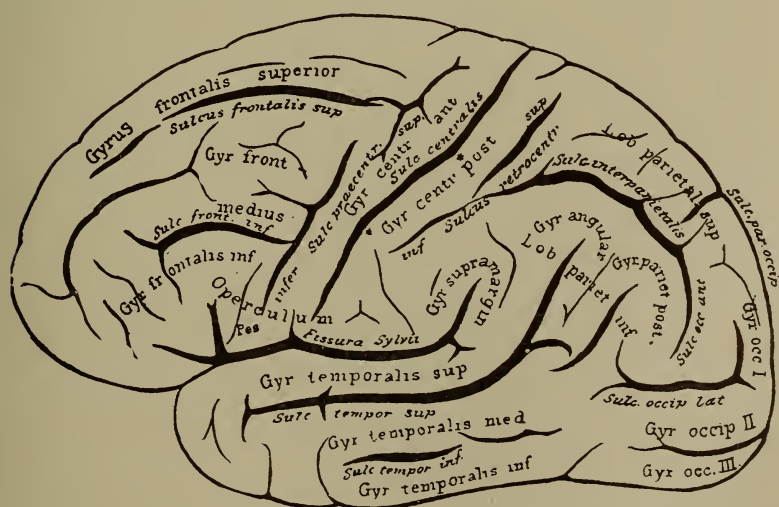
Fig. 39.

From Edinger.
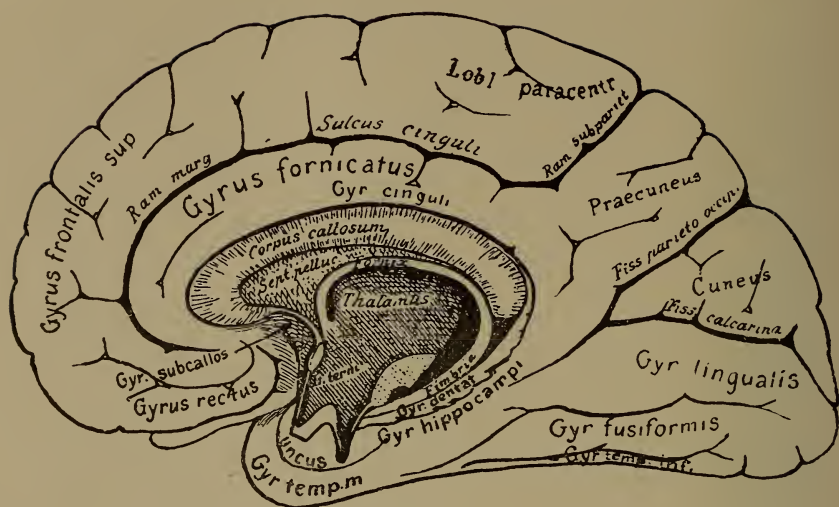
Fig. 40.

From Edinger.

Figures 39 and 40 show the locations and names of the different convolutions, lobes and fissures. The frontal lobe extends as far backward as the fissure of Rolando and downward to the fissure of Sylvius. It extends over and around to the inner face of the hemisphere as far back as the calloso-marginal fissure and an indeterminate line ascending from the latter to the upper edge of the hemisphere.

The parietal lobe is included between the fissure of Rolando in front, the fissure of Sylvius below and a line drawn from the posterior end of the latter to the lower end of the anterior occipital fissure, thence extending to the parieto-occipital notch. On the inner face of the hemisphere the parietal lobe is represented in the precuneus between the ascending part of the calloso-marginal and the parieto-occipital fissures.

The boundaries of the occipital lobe are the parieto-occipital notch above, in front the interparietal and anterior occipital fissures and a line from the anterior end of the lateral occipital fissure downward to the lower edge of the hemisphere.   On the inner face of the latter the occipital cortex is in the cuneus and the lingual lobule.

The temporal lobe includes all of the cortex below the fissure of Sylvius and in front of the imaginary lines already described in bounding the parietal and occipital lobes.   Beneath, it is continuous with the occipito-temporal convolution, and on the inner face, with the uncinate gyrus and the gyrus of the hippocampus.
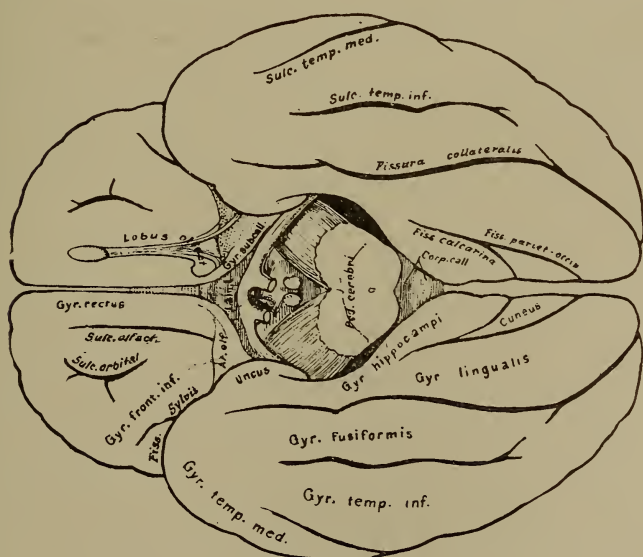
Fig. 41.

Fig. 41, from Edinger, shows the convolutions on the under surface of the cerebral hemispheres.   The convolutions or gyri are marked on one hemisphere, the fissures or sulci on the other.   Their continuance with the inner and outer face of the hemispheres is readily seen.

The cells of the cerebral cortex are of different
kinds and have been depicted as being in several
layers, from four to nine in number according to the
views and interpretation of the observer. The cells
may be classified according to their respective functions
and these are to send out to or to receive impulses
from the periphery (here meaning any part outside
the brain) and to interact among themselves in co-
ordinating or integrating separate impulses or in arous-
ing to activity cells not directly impressed (reproduc-
tion of memories, association of ideas).

The neuraxones from the cells of the motor area
and the fibres bringing sensory impulses to the sensory
areas are called the projection system of fibres; fibres
passing from the cells of one convolution to those of
another in the same hemisphere make up the as-
sociation system of fibres; and fibres that connect the
cells of any cortical part of one hemisphere with the
analogous part of the other are known as the com-
missural system of fibres.

A   motor   impulse   (projection   system)   is
carried by two neurones only, a central and a
peripheral one. The sensory path to the cortex from
the periphery is over two or more neurones. In the
association system there are long tracts as well as
very short ones; thus, the frontal lobe is connected
with the temporal and occipital lobes, the extremes
of the temporal and occipital lobes are connected by
fibre tracts that can be readily demonstrated.

The fibres of the commissural system make up the

bulk of the corpus callosum and the anterior white commissure.

Experimental investigation on the lower animals and accidental ones on man have shown conclusively that certain definite areas of the cerebral cortex subserve certain functions; that when a certain part is excised or otherwise destroyed there follows loss of motor power in a definite part of the body and that if in another animal the same cortical area be irritated there appears motion (spasm) in the part analogous to that paralyzed in the previous experiment. Further, the proof from pathology puts the truth of the doctrine of brain localization beyond question.

To go more deeply into the methods of these investigations or their history is beyond the scope of this book. Their results as at present accepted are shown in Figure 42.

The cortex of the central convolutions of one hemisphere is the motor area for the opposite half of the body and is divisible into three sub-areas, the upper third for the lower limb, the middle third for the upper limb and the lower third for the face (including lips, tongue, larynx, and pharynx).

Finer subdivisions of these sub-areas has been figured by different authors but have not as yet received full acceptance.

The sensory impulses from the periphery are received by the cells in the posterior central convolution and probably also in the cortex extending a little behind the interparietal fissure. It is held by many that

there are cells with sensory function in the anterior central convolution.

The cortical areas for some of the special senses are well known. The visual cortical area is in the occipital lobe and chiefly in the cuneus with some extension to the occipital convolutions on the outer face of the hemisphere.

The auditory area occupies the greater part of the superior and middle temporal convolutions.

The areas for taste and smell are in the convolution of the hippocampus and in the uncinate gyrus (some writers give just the reverse).
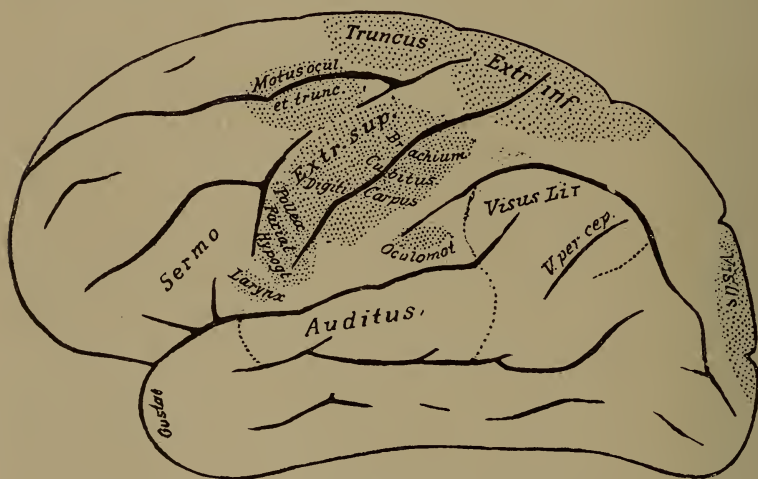
Fig. 42.

Fig. 42. From Edinger (modified). The motor speech area is indicated by the word *Sermo* ; the auditory speech area by the word *Auditus ;* the visual language area by *Visus lit.,* and the visual perception memory area by *V. percep.*
In the figure the tip of the temporal lobe is assigned by Edinger as the cortical area for the sense of taste. In the text an alternative location is given.

It is axiomatic now that when a cortical cell acts for the first time it undergoes some change, that some permanent alteration is thereby made in it and that when it is called upon to act again the individual

is conscious that he has had such experience before. This is the explanation of memory. If the first impression or change in the cell were made under special intensity, or if the action be sufficiently often repeated the individual can at will call up a mental image of the object seen, or a mental reproduction of the sound heard (in the case of such sensory impressions) or can repeat some intricate muscular action. We possess, therefore, the faculty of memory not only for sensation received but for motor impulses sent out.

The child learns to talk by a very slow continuous process lasting during several years. And when it has learned, its faculty of articulate speech is simply the aggregate of all its motor memories of how to arrange its lips, tongue and pharyngeal muscles to produce certain definite sounds. The area for such muscular memories is located in the posterior part of the inferior or third frontal convolution. This is also called Broca's centre, from the name of the physician who first successfully called attention to disease of this area as the cause of loss of the speech faculty without loss of power in the muscles used in speech production.

This result is termed motor aphasia; the subject not only cannot speak, but he also cannot repeat what another has requested him to say, nor can he read aloud.

The areas for sensory memories of language are included in or are near to the sensory areas for hearing and sight.

The area for auditory speech memories embraces the posterior half of the first temporal convolution and a small part of the second. Destructive lesion of this area causes loss of the memories of the meanings that the individual has learned to attach to certain uttered sounds or words. Hence, a person so affected is, when hearing his own language spoken, as one listening to a foreign tongue. He is not deaf, for sounds other than those of language are heard and appreciated as well as ever. The condition is known as auditory or sensory aphasia. In this case he can speak but is apt to use words wrongly; he cannot speak "after" another, nor read aloud correctly, because he no longer has the regulating influence of the auditory speech centre.

Visual memories related to language are those of printed or written words. They are stored up in the cortex of the angular gyrus. Destructive lesion in this area causes loss of the ability to read and the individual is in the position of one who has never learned to read. The condition is known as word blindness or alexia.

An infrequent form termed optical aphasia is that in which the individual while recognizing by sight certain objects (watch, stove, etc.,) and knowing their purpose yet cannot "find" their names, but may be able to do so when aided by the sense of touch or of hearing, from whose speech centres the motor speech memory may be aroused. The lesion is believed to be in the cortex between the angular gyrus and the extremity of the auditory speech area.

The areas for language are marked in Fig. 42, page 252.

Writing from a copy or at dictation may be either lost or retained in motor aphasia; in auditory aphasia the former is retained, the latter lost. As the lesion may not be destructive of a whole area, degrees of loss in the different aphasias must exist and in many cases affected, a complicated symptomatology is presented. As the different speech centres are connected, each with all the others, by association fibres, it is evident that a lesion interfering with conduction along one or more of these pathways must cause disturbance in the inter-relation of those centres among themselves. Finally, lesion in the pathways of conduction of the motor speech centre with the organs of speech, and of the organ of hearing with the auditory speech centre, causes aphasia of a simpler type than in the cortical forms. The making of a schema explanatory of the physiological bases for the production of speech and its understanding, brings into the problem elements that are psychological, which must in any case be influenced and varied by the inventor's self-introspection.

Paraphasia is the faulty use of words by substitution; the individual intending to ask for water, for instance, may utter the word "stable" or some other unrelated word. Conversation becomes almost impossible when paraphasia exists. It is evidence of loss of conduction between the speech centres or of a lesion in the auditory area; it does not occur in subcortical sensory aphasia, and, of course, will not exist in cortical complete motor aphasia.

The terms just used are, cortical, meaning that the actual cortical area is directly affected; sub-cortical, when the pathway between the peripheral organs and the related areas is injured. The term transcortical, as applied to aphasia, means that the pathway from a supposititious "idea" centre (which in turn is used only to represent concretely the summation of all the memories such as the color, shape, weight, temperature, smoothness or roughness, taste, odor, etc., which, taken together, make up the idea of an object), to the motor speech area or from the sensory speech areas to the assumed "idea" centre, is injured.

A "writing centre" in the cortex is not believed to exist.

Voluntary writing is initiated in the young, and in the poorly educated adult, primarily from the arbitrarily assumed "idea" centre *via* the motor speech centre. Thus may be explained the movements of lips and tongue in children when learning to write and also in the adult who rarely writes. In the ordinarily well educated person the writing act is probably dominated by the auditory memories of the words to be written, hence from the auditory speech centre; in those whose intellectual work is predominantly reading, the act of writing is probably immediately governed from the visual centre for language.

The schema, Fig. 43, of Wernicke, and the explanatory table, apply to pure cases of isolated forms. Such cases are not common, and when two or more forms are associated the results of examination of the

patient's powers of language may not be susceptible of satisfactory interpretation.
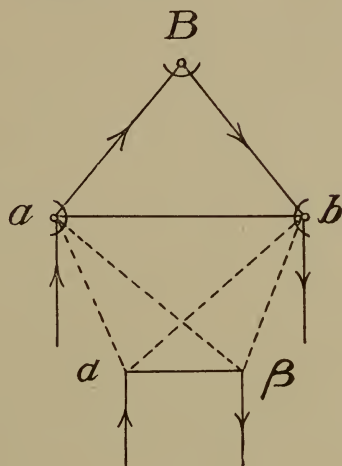
Fig. 43.

Cortical a.—Loss of understanding uttered language
    Cannot speak after another
        "   write at dictation
        "   write voluntarily
        "   read aloud
    Can copy
    Can speak voluntarily but with paraphasia
Cortical b.—Cannot speak voluntarily
        "   speak after another
        "   write from dictation
        "   write voluntarily
        "   read aloud
    Can understand what is spoken
    Can copy
Subcortical a.—Cannot understand uttered language
        "   speak after another
        "   write from dictation
    Can write voluntarily
        " copy
        " speak voluntarily
        " read both to one's self and aloud
Conduction aphasia, between a–b.—
    Can speak voluntarily and after another
        " understand spoken words
        " copy
    Cannot read aloud
        "   write at dictation
        "   write voluntarily
Transcortical b.—Cannot speak voluntarily
        "   write voluntarily
    Can speak after another
        " understand spoken language
        " read understandingly and aloud
        " copy
        " write from dictation
Subcortical b.—Cannot speak voluntarily
        "   speak after another
    Can copy
        " understand spoken language
        " read understandingly
        " write at dictation
        " write spontaneously
        " express number of syllables in a word
Transcortical a–.B—
    Cannot understand words heard
        "   understand what he reads
    Can speak after another but without understanding
        " read aloud without understanding
        " write voluntarily but with paragraphia
        " write at dictation
        " speak voluntarily but with paraphasia
        " copy

a represents the auditory cortical speech area; *b*, the motor cortical speech area. *B*, the assumed idea centre. The dark lines below *a* and *b* are the pathways from the auditory organ, and that to the organs of speech respectively. The arrow - heads show the direction of conduction.

The association fibres between the auditory speech area and the motor speech area are represented by the line from *a* to *b*. Interruption in it causes conduction aphasia.

Destructive lesion at *a* causes cortical auditory aphasia.

Lesion at *b* causes cortical motor aphasia.

Lesion in the fibres (dark line) below *a* causes subcortical auditory aphasia.

Lesion in the fibres (dark line) below *b* causes subcortical motor aphasia.

Lesion between *a* and *B* causes transcortical auditory aphasia.

Lesion between *b* and *B* causes transcortical motor aphasia.

Alpha represents the cortical visual language area; beta represents the motor area for right hand. In reading aloud the visual area arouses the auditory area *a*, and this, in turn, the motor speech area *b*. Understanding what is read is by continued transmission from alpha to *a* and thence to *B*.

Writing at dictation is *via* *a*, alpha, beta.

Copying needs only the visual language area and the motor area in left hemisphere for the hand, and association fibres, thus alpha—beta.

The visual memories of objects perceived are stored up in the cells of the angular gyrus, but as it is next to the supramarginal gyrus, disease of one area often affects the other. Hence, with loss of the power of reading there is frequent association of loss of the recognition of familiar objects. The latter condition is known as **apraxia**; in it the patient is unable to recognize by sight objects of every-day life, but may be able to do so through uninjured areas for other modes of recognition. Thus, a watch being unrecognized by sight may be at once correctly perceived when it is placed near the ear and its ticking heard.

Lesion of the fibres beneath the angular gyrus has been oftener noted than lesion in the cortex.

The various sensory and motor faculties connected with language are in the left cerebral hemisphere in right-handed persons; in left-handed persons they are in the right hemisphere. In either case after destruction of an area the corresponding area of the opposite hemisphere soon learns to act as substitute.

The visual memories of objects (perception) appear to be represented in the angular gyri of both hemispheres. (See Fig. 42, page 252.)

Aphasia must be distinguished from dysarthria or anarthria, in which the muscular activities involved in producing articulate speech are impaired or lost as a result, commonly, of degenerative disease in the medulla (*vide* bulbar paralysis).

Aphasia is only a symptom, most frequently from

lesion in the affected area, or the many association paths from one area to another. It occurs also as a result of toxic influences (infectious diseases, uræmia, etc.), from trauma (depressed fracture of the skull affecting one or more of the areas related). It also may appear in conditions of exhaustion and after fright; in the latter case if complete motor aphasia exist it may be difficult to distinguish it from hysterical muteness.

Treatment. When an organic affection is the cause of aphasia the treatment must be directed to such disease. When functional only, the symptom aphasia will be taken into account. ·

Our materia medica contains very little of purely aphasic symptoms of any one variety. Motor aphasia is found under *Chenopodium anthelminticum;* paraphasic symptoms are given under *Natrum mur., Calcarea carb.,* and *Lycopodium;* alexia, under *Lycopodium, Hyoscyamus* and *Plumbum,* and optical aphasia under *Baryta acetica.* Some of the symptoms given are possibly due to general mental weakness and the differentiation of remedies for the varieties of aphasia must be made now from clinical experience.
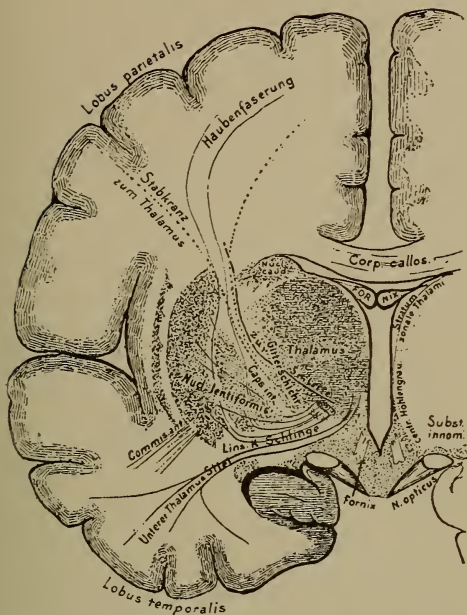
Fig. 44.

Fig. 44, from Edinger, shows a perpendicular transverse section of the brain through the optic thalami. The left optic thalamus is shown separated from the right one by the third ventricle. The positions of the nucleus caudatus and nucleus lentiformis and the relation of the three bodies to the internal capsule are evident. To the outer side of the lenticular nucleus is seen the cortex of the island of Reil at the bottom of the so-called fissure of Sylvius. The positions of the corpus callosum, fornix and optic nerves, all cut through, are indicated. The drawing is largely schematic.

Fig. 45, from Edinger, is a horizontal section of the brain. The various parts are indicated by their names. *Balken* = corpus callosum.
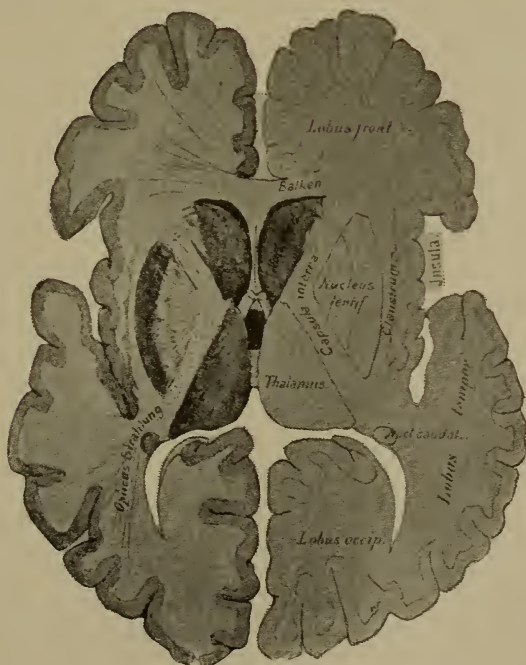
Fig. 45.

Anæmia of the Brain.

As an independent primary condition anæmia of the brain cannot exist, but is found as a secondary state, the result of vascular disease or of cardiac weakness, or as part of a general anæmia.

In its acute and usually temporary manifestation the symptoms may be alarming to lay observers. This is termed syncope or a fainting fit, and is commonly caused by fright or similar emotional shock, which by influencing the heart results in lessening or stopping the flow of blood to the brain. The symptoms are mental confusion, loss of vision, vertigo, nausea, loss of power over the limbs, so that the patient falls and then remains apparently unconscious, or nearly so, for a short time, varying from a minute or a few minutes to a considerable fraction of an hour. In less severe cases the above-mentioned symptoms are less in degree.

In cases of chronic cerebral anæmia there are drowsiness, which is replaced by insomnia on lying down (effect of position), mental depression, apathy, with inability or aversion to work, attacks of fainting, weakness of memory, etc. All of the symptoms except insomnia are relieved by lying down.

The prognosis of the acute condition, especially as seen in young persons, is favorable, and there is no danger to life even in a prolonged syncope when it is not dependent upon an excessive loss of blood or pronounced cardiac disease.

In chronic cerebral anæmia the prognosis is that of the underlying cause.

Treatment of an attack of syncope should always include placing the patient in a horizontal position without pillow for the head, and stimulating reflexly the respiratory and cardiac centres by dashing cold water on the face and chest.

Hyperæmia of the Brain.

When, from any cause, the flow of blood into the cranial cavity is in such amount or at such rate that the veins cannot carry it off in equal degree there is present an excess of arterial blood; and when the capacity of the veins is for any reason increased in relation to a normal influx of arterial blood, there will be an excess of venous blood. Both conditions are hyperæmias, the former being termed active, the latter passive. Active or arterial hyperæmia may be produced by strong emotion, such as a fit of anger, by the action of certain drugs, such as alcohol, nitrite of amyl, nitroglycerine, etc., by a cold bath, etc. Passive or venous hyperæmia is usually a continuous state dependent upon some mechanical obstruction to the venous efflux from the brain, such as tumors of the neck pressing upon the jugular veins, certain diseases of the heart, emphysema of the lungs, etc., but a passing hyperæmia of this kind follows muscular effort in which the diaphragm assists, as in the case of lifting heavy weights, straining at stool, etc.

Repetition of the causes of arterial hyperæmia re-
sults in a more permanent dilatation of the arterioles
and this in turn gives rise to retardation of the blood
current with consequent lessened rate of metabolism
within the brain, an engorgement hyperæmia, yet with
many of the symptoms seen in states of cerebral
anæmia.

The symptoms of acute arterial hyperæmia of the
brain are a rising of heat to the head and face, with
a sense of distension, pulsation felt in the carotids
and head, headache, at times vertigo or even partial
obscuration of consciousness. The symptoms are
aggravated by stooping, coughing, lying down, etc.
Such acute congestion may be accompanied by a rise
of body temperature, and cannot be distinguished
from the early stage of a meningitis. Passive hyper-
æmia from venous obstruction below has similar symp-
toms, but usually of less intensity and without the
marked pulsation seen in the active arterial type. In
continued or chronic states of engorgement hyper-
æmia due to loss of tonicity of the vessels' walls,
there are sleeplessness with more or less drowsiness
when up and about, weakness of the mental powers,
especially when concentration of mind is required,
headache, with emotional depression or even unmotived
anxiety or morbid fears. This condition is cerebral
neurasthenia.

Prognosis in cases of active or arterial congestion is
good, except where cardiac disease seems to be the
cause; in the form due to venous obstruction the

prognosis is that of the causal condition; in the form due to vascular dilatation the prognosis for cure will depend on the length of time the condition has existed, upon the underlying cause (alcohol, tobacco, etc.). In cases of recent origin or where reformation in the mode of life can be established, a cure may reasonably be expected. Oppenheim considers that masturbation plays a predominant *rôle* in causing dilatation of the cerebral blood vessels.

The treatment of cerebral hyperæmia must be by remedies chosen with recognition of the causal condition if such can be found, and the accompanying symptoms. As a symptom, a congestive cerebral state can be produced by very many drugs; in the Symptom Register to Allen's Encyclopedia 52 drugs are given under the rubric Congestion of the Head. The remedies most often indicated in the condition are *Belladonna, Hyoscyamus, Melilotus, Opium, Lachesis, Calcarea carbonica, Ferrum phosphoricum, Nux vomica* and *Veratrum viride. Dirca palustris* and *Fagopyrum esculentum* have been effective in the writer's hands. In passive hyperæmia *Chloral hydrate* 30, *Baryta carbonica, China, Silicea, Piper methysticum* and *Amyl nitrite* in potency have been of great value. In any case the accompanying symptoms should decide the choice of the remedy.

Cerebral Meningitis.

Inflammations of the meninges of the brain are classified as pachymeningitis and leptomeningitis according

to the membrane chiefly affected, the dura, or the pia and arachnoid.

External pachymeningitis (dura mater) occurs at times as a result of disease in the skull bones. Its symptoms are the general ones, fever, headache, perhaps delirium or even convulsions and its relief or cure depends on removal or lessening of the externally acting cause.

Internal pachymeningitis is hæmorrhagic in its nature, the hæmorrhage occurring in successive outbreaks. The effused blood becomes organized and forms layers of connective tissue which are readily separable on post-mortem examination and are studded, especially the innermost ones, with minute new hæmorrhages.

The thickening thus formed acts as an extra-cerebral tumor and upon its location will depend the special symptoms. It occurs in chronic alcoholism, in dementia paralytica or other chronic wasting disease of the cerebral cortex, as well as in chronic kidney disease. It is stated to have been observed in individuals of the hæmorrhagic diathesis and during the course of pernicious anæmia, leucæmia, scurvy, etc.

Light cases may show only the symptoms of some general affection of the brain, but in a severe case there is marked cerebral excitation similar to that of delirium tremens, or a unilateral epileptiform attack. This may be followed by a comatose state that may last for days or even weeks (Oppenheim). With the usual signs of increased brain pressure the first attack

may end fatally and then cannot be distinguished from
an apoplexy, but if recovery occurs it is apt to be
complete. The diagnosis is difficult and at first may
be impossible; the chief point is the recurrence of
symptoms of cortical irritation in an alcohol *habitué*,
an old person, or after external injury to the head.

Leptomeningitis or inflammation of the pia (and
usually the arachnoid) occurs in three or four distinct
forms—the purulent, tubercular, syphilitic, and an
epidemic form known as cerebro-spinal meningitis.

Purulent leptomeningitis is caused by some pyogenic
micro-organism; thus the disease is an outgrowth of
some infectious process in a part of the organism other
than the brain. Thus pyæmia, septicæmia, pneumonia,
scarlet fever, and similar diseases may give rise to
an infection of the meninges *via* the blood vessels.
Direct invasion, by way of the sheaths, along the out-
side of nerve trunks, or of vessels from suppurating
neighboring structures is not uncommon. Injuries to
the head resulting in an open wound may give access
even through unbroken bone to the germs (v. Berg-
mann).

The disease-process affects chiefly the convexity of
the cerebral hemispheres, but in many cases the mem-
branes at the base are also involved. Hyperæmia of
the pia-arachnoid, then thickening and turbidity and
next the appearance of pus, mostly aggregated along
the fissures, is the order of succession. The cortex
may become involved, small hæmorrhages, foci of sup-
puration and an interstitial œdema being present; or

the ventricles may show inflammation of their lining, often with a sero-purulent fluid within or even a real hydrocephalus.

Fever in the beginning and of atypical course, with or without a chill, violent headache, continuous but with times of aggravation, obscured consciousness with delirium or sopor are common symptoms, while during the delirium and even in sopor, evidences of the existence of severe headache are present. The pulse is often irregular, bowels confined, abdominal walls retracted and extreme hyperæsthesia both of the periphery and of the special senses is present. Convulsions may occur, either general or limited to one side or to a single limb.

When the disease-process affects the basal membranes the symptoms are those of compression or irritation of the cranial nerves, such as paralyses or spasm of ocular muscles, inequality of pupils, absence of the light-reflex, optic neuritis, facial paralysis or facial spasm, gnashing of the teeth or even trismus.

Perhaps the most diagnostic symptom of meningitis of any kind that has invaded the posterior fossa of the skull is the retraction of the head, with rigidity of the neck, often accompanied with rigidity of the limbs. In the later stages coma with loss of the deep reflexes comes on, together with irregular and rapid pulse, rapid and sometimes irregular respiration or respiration of the Cheyne-Stokes variety. Death occurs in the increasing coma or in convulsions.

The disease is always acute in character and may

end fatally in a few days, but more commonly it lasts from one to two weeks. When coma comes, a fatal ending may be predicted (Oppenheim).

Recovery has been observed, in one case of the writer's with serious damage to the cranial nerves and the mental powers.

The prognosis is always grave.

The diagnosis will be made from the rapid occurrence of cerebral symptoms in individuals suffering from septic or suppurative processes in other parts, or from infectious diseases. But these diseases themselves may begin with cerebral symptoms that later clear up.

Tubercular meningitis, like purulent meningitis, is to be considered a secondary disease, but, unlike the latter, is always due to the invasion by the same bacillus or to the effects of its toxine. Its victims are never those in good health, although in some instances it may appear to be the primary disorder, but are pale, weak, ill-nourished, "scrofulous" individuals, already affected with some form of tuberculous deposit.

It is mostly seen in children between the ages of two and twelve, but occasionally in the adult.

The disease is characterized by a long prodromal stage, as might be inferred from the kind of bacillus at work. The tubercle bacilli being carried *via* the blood vessels to the meninges, settle thereon and, as a result of their presence and irritative action, inflammatory exudative changes in the membrane occur,

with subsequent changes known as the eruption of
miliary tubercles, in which the characteristic bacillus
tuberculosis is found. The process is mostly at the
base, but usually extends to the spinal membranes.
Along the fissure of Sylvius and other large fissures
on the convexity, tubercles and exudation are found,
but in less amount.

The prodromal symptoms are those of general mal-
aise. The child becomes languid, refuses to play,
becomes irritable or moody, with restless nights or
even sleeplessness. Headache comes on, at first light
and temporary, later severe and continuous, yet with
periods of violent exacerbation, and vomiting without
cause and projectile in character, constipation and
occasional rises of temperature. Intermittency of these
symptoms is to be expected during its early or pro-
dromal stage, which may last during three or four
weeks or may extend over a much longer period.

When the disease has, so to say, settled, and the
meningitic process has begun, the headache becomes
continuous and intense, delirium appears, with great
restlessness, fever of an irregular type, pulse showing
the same peculiarity, but more often slow, partial
or general convulsions, gnashing of the teeth; the
sleep, such as it is, is interrupted by a sudden pierc-
ing scream (cri hydrencéphalique).

The symptoms due to irritation or pressure at the
base are those of interference with the cranial nerves
there, especially the ocular nerves. Inequality of the
pupils with dilatation (less often contraction), with

loss of the light-reflex, paralytic strabismus, associated deviation of both eyes to one side and even optic neuritis are symptoms to be looked for. Symptoms due to implication of structures in the posterior fossa are retraction of the head, with rigidity of muscles at the back of the neck, with extension to the back, and even the limbs.

The delirium passes into sopor and then into coma, deceptive remissions being not infrequent; the coma becomes more profound, the respiration irregular, assuming at times the Cheyne-Stokes type, a general paralytic state of the muscles replaces rigidity, and with a rapid rise at times (104° or 105° F.), or lowering of temperature (96° F., or even lower) death ensues.

The lateral ventricles generally contain an increase of fluid, turbid or at times sanguinolent in character; this state of **hydrocephalus internus** may be extensive enough to affect, by pressure, the cortex.

Diagnosis is to be made chiefly by the history (tuberculosis elsewhere), the age of the patient, and the early cerebral symptoms coming on in spells either singly or grouped.

Prognosis is bad, but cases have been reported in which cure has resulted; here the question of diagnosis must be brought forward.

Chronic meningitis as a manifestation of chronic alcoholism appears predominantly on the convexity, while when the result of the syphilitic process it affects chiefly the base. The symptoms in either case

are those of meningitis of the related type, although of less intensity, but the diagnosis of chronic meningitis, except when the above-named etiological conditions can be shown to exist, is practically impossible during life. Cases are said to have occurred as the sequel or continuation of the acute cerebro-spinal meningitis or of insolation.

Epidemic cerebro-spinal meningitis is a disease due to a germ similar to if not identical with the pneumococcus. The disease occurs in the temperate zones, especially during the winter and spring, and in crowded dwellings, and chiefly among children and youthful individuals. It appears epidemically, but its distribution seems to depend in some degree upon climatic or other similar conditions, since it is a rather common affection in some parts of the United States, while it is rare in other parts in nearly the same latitude. Sporadic cases, of course, can occur.

The invasion by the disease is rapid and in some cases almost sudden. The early symptoms are general malaise, chilliness, restlessness, pains in the back and back of head and at times in the extremities, and the evidences of a severe illness are apparent. Next, vomiting, hypersensitiveness to noise, light and touch, with delirium and fever, come on. The temperature is variable, rarely goes beyond 104° F.; the pulse is rapid and also variable. Of diagnostic value is the appearance of herpes labialis in the early days of the disease. Retraction of the head, stiffness of the neck muscles, and contraction of the muscles of the back,

even to the production of opisthotonos, are symptoms
that come on within the first few days, in varying
degree. In most cases, some form of eruption appears
on the skin, such as erythema, urticaria, roseola,
purpura, the latter being of great diagnostic value.
General convulsions may occur early in the disease
and may recur.

In unfavorable cases symptoms of paralysis with
coma in the course of a few days or by the end
of a week, and paralytic phenomena involving some
of the cranial as well as the spinal nerves, are to be
expected; these are myosis or other alteration of
pupils, paralysis of muscles of the eyeballs, nys-
tagmus, optic neuritis, partial or complete deafness,
monoplegia, hemiplegia, or even paraplegia.

In severe cases the disease runs a very rapid
course, death occurring within a few days; in cases
of moderate severity the disease may extend over
two or three weeks. The mortality varies in dif-
ferent epidemics, in some 75 per cent. having been
noted, the average being 57 per cent. (Oppenheim);
towards the end of an epidemic lighter cases are in
the majority.

Pathologically the disease process is an inflamma-
tion with fibro-purulent exudation in the pia extend-
ing over the convexity and base of the brain, and
also to the membranes of the cord with special ac-
tivity around the lumbar enlargement. The cranial
nerves, especially the optic, third and eighth are infil-
trated or embedded, and the latter nerve may be a

pathway for extension of the inflammation to the inner ear and middle ear.

Diagnosis is not difficult in an ordinarily well marked case if similar ones exist in the immediate neighborhood (epidemic), but in sporadic cases the affection has to be differentiated from other forms of meningitis (tubercular) and from other infectious diseases, especially pneumonia, which, indeed, may be associated. An absolute diagnosis may be made by aspiration of the spinal canal in the lumbar region, the aspirated fluid showing the presence of the meningococcus intercellularis, or of the pneumococcus, the latter apparently being the more virulent. Herpes labialis does not occur in tubercular meningitis.

Sequelæ are blindness, deafness, chronic headache, chronic spinal meningitis, muscular incoördination, and sometimes paralysis. Blindness and deafness improve spontaneously in some cases, but in most remain.

The treatment of any form of cerebral meningitis must at first be largely symptomatic. If any causal indication exist it should be heeded ; hence, in cases of traumatic origin *Arnica* will naturally be first thought of; if from exposure to excessive heat, *Glonoine*. In general, the symptoms in the beginning will call for *Belladonna, Bryonia, Gelsemium* or *Aconite.* Later, if symptoms of cerebral irritation are pronounced, *Cantharis, Hyoscyamus, Cicuta virosa, Cimicifuga, Veratrum vir.* or *Zincum* may be required.

If effusion is evident from the signs of increasing brain pressure, *Apis*, *Helleborus*, *Opium* or *Digitalis* may be required. *Bryonia* is highly regarded by different observers when there is excessive pain. *Iodoform* 6x or higher, has, in the writer's hands, been of signal service in chronic meningitis. *Cuprum aceticum*, *Zincum met.*, *Crotalus horridus*, *Rhus tox.*, *Calcarea carb.*, *Lycopodium* and *Sulphur* have been reported as curative or beneficial. In all cases the symptoms should be the guide to the remedy. In tubercular meningitis the brain symptoms will not lead to a selection of a remedy for the real disease. The fatality of this form of meningitis will make it advisable to use *Tuberculinum* in a high dilution.

Chronic hydrocephalus is, in the vast majority of the cases, a condition beginning in intra-uterine life. It may have advanced at term to such degree as to cause difficulty in delivery or at such time it may be latent and only develop within a few weeks or months.

The condition is an accumulation of fluid within the ventricles of the brain and is due to occlusion of the openings of communication between the general ventricular cavity and the cavity of the arachnoid, or between the cavities themselves. The development of the latent disorder may not occur until set in action by some accident, trauma, or some trifling intercurrent affection.

The manifestation of the disorder is a gradually progressing enlargement of the head. The enlarge-

ment is in all directions, forwards, backwards, later-
ally and even upwards, but here the yielding of the
fontanelles and opening of the partly closed sutures
allows greater horizontal distension. The face, below
the roofs of the orbits, of course, shows no change,
and there is a marked contrast of small face and large
head, the general outline being triangular when viewed
from the front, while from above the outline is more
or less round. Increasing pressure of the fluid within,
results in a thinning of the skull bones, which in ad-
vanced cases can be felt.

Cerebral symptoms appear; impeded development
of the brain results in retardation of mental develop-
ment as well as of muscular growth and control. The
child cannot learn to walk, at times cannot sit up
on account of the weight of the head, does not learn
to talk. The cranial nerves are affected by the pres-
sure, and strabismus and, occasionally, optic nerve
atrophy are seen.

The general state of the patient shows cerebral
irritation; the child is poorly nourished, generally
wretched and unclean. Paresis of the lower limbs,
with increased tendon reflexes, is not uncommon.

The prognosis is bad, most of the cases dying
within two or three years. Lighter cases may grow
up, with development of mental and physical powers
to a considerable degree, the enlargement of the
head remaining.

The diagnosis is to be made from rickets, or in
later years from acquired hydrocephalus, the result

of pressure by a tumor in the posterior fossa upon the vena magna Galeni, obstruction of the aqueduct of Sylvius, etc. In rickets the shape of the head is box-like, the fontanelles are not prominent, there are other evidences of the disease and cerebral symptoms are absent.

The treatment of chronic hydrocephalus should be by the employment of our deeply acting remedies, such as *Calcarea carb.*, *Calcarea phos.*, *Silicea*, *Sulphur* and *Baryta carb.* Rapid improvement must not be expected. In one case of the writer's after several months' treatment with some of the remedies just mentioned, marked change for the better appeared on giving *Helleborus* 6.

Abscess of the Brain. The suppurative process in the brain requires the presence of a pyogenic microbe. This may be introduced from without *via* a scalp wound (without any fracture of the underlying bone), or from within by direct invasion from some suppurating process in the neighborhood (chronic purulent otitis media), or by means of the blood vessels, from suppurative foci in distant parts (abscess of the lung, empyema, etc.).

An acutely occurring abscess is chiefly seen as a result of trauma, and then affects the convexity, the symptoms being, like those of meningitis, mental confusion, delirium, fever of irregular type, with severe headache. If the motor area be involved, there will develop hemiplegia or monoplegia, preceded by epileptic convulsions.

Acute symptoms may be the culmination of the chronic process.

The other forms of brain abscess are of gradual development, and may remain latent for months or even years, the period of latency being characterized by irregular outbreaks of cerebral symptoms, with increase of temperature; the acute episodical manifestation disappears, and the patient returns to a state of health, which, in turn, is again invaded by a recurrence of the previous symptoms.

Headache, often of a severe type, is a frequent symptom; it is not seldom located at or over the site of the abscess. Vomiting is frequently seen; optic neuritis, while not uncommon, is yet less often observed than in brain tumor. The pulse is slow generally, but at times is accelerated.

The cerebral hemispheres are much oftener the seat of abscess than the cerebellum, while abscess of the pons or medulla is rare. In the cerebrum the temporal and frontal lobes are more often the seats of abscess, and if this is very extensive and deep, many of the underlying tracts will be invaded, giving rise to sensory aphasia (often misinterpreted as evidence of mental weakness — Oppenheim), injury to the motor tract, hemianopsia; if in the occipital lobe, hemianopsia is to be expected.

Brain abscess, if otitic in origin, will occur in either the temporal lobe or the cerebellar hemisphere, according to the location of the original affection on the anterior or posterior part of the petrous bone.

Abscesses of distant origin (suppurative processes in the lungs, empyema, etc.) are located most often in the region supplied by the Sylvian artery; pyæmia frequently causes multiple brain abscesses.

The diagnosis of abscess of the brain will rest upon the existence of some adequate cause, trauma, suppurative ear disease, etc., but tumor may occur after trauma. The recurrence of cerebral symptoms in attacks often ushered in by chills, the wide temperature range, the non-implication generally of the basal cranial nerves (except the optic, and occasionally the olfactory) are cardinal points. Acute abscess may end fatally before the specially diagnostic symptoms appear, and then the diagnosis from acute meningitis may be impossible. Meningitis is not infrequently set up by suppuration within the brain, while sinus phlebitis may occur from extension of the original ear affection.

The course of acute abscess, or the terminal attack of the chronic affection varies. When traumatic, the fatal termination is delayed more than when due to ear disease. In general, the time limit is from a few days to a very few weeks. Death occurs from coma and exhaustion, or from the breaking of the abscess into a lateral ventricle or externally, with consequent apoplectic or convulsive symptoms, with coma.

The treatment of abscess of the brain is purely surgical.

Apoplexy.

Cerebral hæmorrhage. A blood vessel ruptures under the pressure of the blood within it, either because of congenital or acquired weakness of its walls or from abnormal increase of the blood pressure, whether from kidney disease or cardiac hypertrophy or both.

The occurrence of intracerebral hæmorrhage in the infant during the process of parturition may be considered as mechanical, owing to the prolonged or severe compression to which the yielding skull is exposed. Towards middle life the arteries manifest a degenerative change known as arterio-sclerosis, and this increases in degree with advancing years, being greater in some individuals than in others. Such degeneration is hastened by certain diseases, or by long-continued poisoning by certain substances (chronic kidney disease, the long-continued use of alcohol, etc.), while the hæmorrhagic diathesis, leucocythæmia, pernicious anæmia and syphilitic disease of the blood-vessels within the skull cavity, are more direct causes of hæmorrhage.

The influence of heredity is seen in the occurrence of cerebral hæmorrhage during several successive generations, either through inherited weakness of the vessels themselves, or tendency to diseases causing the weakness. Miliary aneurisms situated on the circle of Willis or the arterial

branches arising therefrom, are frequently the sites at which hæmorrhages occur.

Given weak arteries with spots of advanced degeneration and aneurismal protrusions, any sudden increase of blood pressure, as in physical exertion, strong emotion, after a heavy meal, etc., may determine a break in a blood vessel; but, without this, the time must come when the vessel will finally yield under the ordinary blood pressure, and it may be, as is not infrequently the case during sleep.

Prodromal symptoms are rather rare; when present they consist of vertigo, pressure in the head, cardiac anxiety, paræsthesia of one side, perhaps some mental confusion and difficulty of articulation, but such symptoms are not necessarily prognostic of an oncoming hæmorrhage.

The attack comes on suddenly and in most cases during apparent good health, the patient falls to the ground and is at once comatose; the face is turgid, red or purplish; all the reflexes are gone, including the pupillary and corneal reflexes; the pulse is full, strong, and may be slow or normal or accelerated in rate; the skin is usually moist.

The respirations are slow and stertorous and with each expiration the cheeks puff out. Urine and fæces may pass involuntarily; the former generally contains some albumen. The limbs are entirely relaxed, yet when lifted and let drop a difference may often be observed, those of the paralyzed side drop like lead,

those of the other side sink rather than drop, owing to some muscular tonicity remaining.

The temperature may fall a degree or two during the first half day, then it ascends to the normal and may even rise beyond it, while the surface temperature on the paralyzed side is a degree or two higher than on the normal one. Subnormal temperature, if considerable in degree or long continued, is of evil omen.

Should the case progress to a fatal termination, there will be generally a rapid rise of temperature, disturbance of respiration, which may take on the Cheyne-Stokes rhythm, rapid pulse; if the end be delayed beyond a few hours, hypostatic engorgement of the lungs is apt to occur, death following within a day or two. In more prolonged cases the coma may lessen, the corneal and swallowing reflexes return, but the reactive inflammatory process around the effused blood introduces a fresh element of disturbance, delirium often coming on and lasting until death, or being replaced by sopor, coma or other symptoms with lethal ending.

In favorable cases coma lessens within a few hours, a soporous condition serving as transition stage before even temporary consciousness returns, the deep reflexes appear gradually, movements of the unparalyzed limbs occur, as during restless sleep, and when the patient emerges into consciousness there is found some emotional or mental weakness.

The most marked effect of cerebral hæmorrhage

is hemiplegia, face, arm and leg being affected, but the distribution of the facial nerve to the frontalis, corrugator supercilii and orbicularis palpebrarum muscles are usually not implicated, because the central neurones for them are not in the pyramid tract. The hypoglossal distribution is involved in the paralysis, hence the unopposed action of the genio-glossus of the sound side causes deviation of the organ to the paralyzed side when the tongue is protruded. Implication of the lower face muscles is shown by asymmetry of the angles of the mouth, the cheek of the affected side being lower and that of the sound side being higher, owing to the unopposed tonicity of its muscles.

Some degree of anæsthesia is noticed in many cases in the paralyzed parts, and the cremasteric and abdominal reflexes are absent on the paralyzed side. Within a short time the knee-jerk of the affected side becomes exaggerated. Later, ankle clonus can often be obtained. With right - sided paralysis some kind of aphasia, motor or auditory, is to be expected; it is often temporary.

The symptoms following a stroke of apoplexy are classed as temporary, and permanent; the former are also called indirect, because they are not from the direct destructive influence of the lesion which causes permanent effects, since a destroyed nerve fibre in the central nervous system cannot be replaced or restored. Many symptoms are thus indirect. Some amount of voluntary control reappears in the af-

fected limbs; the face loses most of its asymmetry; the leg recovers its powers to a large degree; the arm, while not so helpless as at first, improves in only a slight degree.

Rigidity of the muscles of the affected limbs comes on in some cases early; that is, within a day or two; it may pass away soon or stay for a week or two. But between two and three weeks after the seizure there occurs the late rigidity with contractures. The latter are characteristic when fully developed, the fingers being flexed, the hand flexed on the arm, the forearm flexed and pronated, the arm adducted. The lower limb is in the extended position and when the patient is able to walk gives rise to the peculiar gait of the hemiplegic, for as the patient is able to flex the limb at the knee but slightly, he must, in order to progress, lift up that side of the pelvis (and with it the whole of the paralyzed limb) by contraction of the trunk muscles of the opposite side, at the same time turning the body on the sound leg. Hence he describes on the ground an arc of a circle with the paralyzed member, or, as the Germans put it, "he mows the ground."

If, after three weeks or so, no contractures appear (and no exaggeration of the deep reflexes), complete restoration of voluntary power may be expected, the paralysis in such cases having been owing to indirect influence (pressure) and not to destructive action of the effused blood upon the motor tract.

The muscles of the paralyzed parts waste somewhat,

but generally it is the wasting due to non-use, though in some cases the wasting is too rapid to be thus explained and must be attributed to the cerebral cause; what part of the brain it is that has such trophic influence is not known (Goldscheider). The electrical reactions are normal or nearly so, the re-action of degeneration never occurring. The muscles that act in association on the two sides are not affected, but the trapezius at times shows some weakness in its upper part. The affected limbs are more or less cyanotic, are often somewhat swollen, while the palm of the contracted hand is moist from retained per-spiration.

As late symptoms, secondary to hemiplegia, are seen at times certain associated movements in a para-lyzed limb when its fellow of the opposite side acts, and also posthemiplegic tremor, occasionally of the intentional variety. In children choreiform action, or athetosis (a continuous writhing movement of fingers and hand, or of fingers, hand and arm, occasionally of toes and foot) is present in many cases.

When the site of cerebral hæmorrhage is in the neighborhood of the large basal ganglia and the motor tract, hemiplegia must result, but hæmorrhage may take place in any other part of the brain and then the symptoms will vary accordingly (*vide* Figs. 44 and 45).

Hæmorrhages upon the cortex (vessels of the pia) must be very extensive to cause a hemiplegia, since the whole motor area would have to be subjected to

the effused blood. As a matter of fact, complete hemiplegia from hæmorrhage upon the cortex is rare, while a partial hemiplegia, especially of one limb or part, and begun with spasm, points to the cortex as the site of the lesion.

The centrum semiovale is the site of hæmorrhage less often than the internal capsule and basal ganglia; the nearer to the cortex it is, the more nearly do the symptoms resemble those of the cortical lesion—and the deeper the hæmorrhage, the more closely do they approach those of typical hemiplegia.

Hæmorrhages into the pons are less frequent than those of the cortex. If in the upper two-thirds, a typical hemiplegia may be caused; if in the lower third, a crossed hemiplegia will result—i. e., paralysis of the limbs of the side opposite the lesion, the face being paralyzed on the same side as the lesion. When the hæmorrhage is from a median branch of the basilar artery, as it most often is (Gowers), its location is central, the apoplectic attack is ushered in generally with convulsions that are bilateral mostly, while all four limbs may be paralyzed. In pontine hæmorrhages the danger of death is very great.

At times hæmorrhage in the neighborhood of a lateral ventricle breaks its way into the latter and from it the whole ventricular system may be invaded. The result will be a distinct and alarming aggravation of symptoms already present; coma deepens or returns, the unaffected side becomes paralyzed and convulsed or even general convulsions may occur, tetanic rigidity

being one mode of the latter. The prognosis becomes absolutely hopeless, death occurring within a day or two.

The sensory disturbances during an apoplectic attack already mentioned will, of course, be more marked the greater the invasion of the posterior part of the posterior limb of the internal capsule, and may be permanent; pain may exist in the paralyzed limbs, either continuously or as painful hyperæsthesia on being touched.

Conjugate deviation of the eyes and head to one side is of some diagnostic value. If they are directed away from the paralyzed side it means a cerebral paralyzing lesion or a pontine irritative one; if they are towards the paralyzed side, there are conditions of irritation in a cerebral lesion (cortical or possibly ventricular hæmorrhage without irritation), or the lesion is in the pons and is paralyzing.

The fate of the blood effused in cerebral hæmorrhage is, first, coagulation; next, breaking down of the constituents of the clot; then, absorption. Meanwhile, the surrounding brain tissue sets up protective inflammatory changes in proliferation of glia cells and connective tissue, forming a capsule within which further absorption of the broken-up clot goes on, until finally the latter has disappeared and its place is taken by serous fluid, the result being a hæmorrhagic cyst. In the case of quite small hæmorrhages, the walls approximate and the space is bridged across by connective tissue, whose subsequent contraction results

in the formation of an apoplectic cicatrix. Either process requires considerable time, the formation of a hæmorrhagic cyst requiring at least three or four weeks (Oppenheim).

Apoplexy from Vascular Occlusion.— Acute Encephalomalacia.

When an artery is occluded in the brain, the tissues depending on it for their blood supply must die for want of nutrition, there being in the brain but little vascular anastomosis. The occlusion may be either from an embolus or a thrombus.

An embolus, being a detached mass of fibrin in the arterial current, gets to a point beyond which in the narrowing vessel (usually where a branch is given off) the blood current cannot carry it.

The source of the embolus is in most cases the heart itself, which, when diseased as by endocarditis, or with valvular trouble, has deposits within it of coagulated fibrin. Of the latter a fragment, if detached, is sent out in the blood current and is arrested as described above, in some small vessel. Emboli are at times carried by the blood-current from a thrombotic deposit in a diseased large blood vessel, or enter the heart from the venous side in cases of pulmonary gangrene, ulcerative bronchitis, etc.

Thrombosis, being a deposit chiefly of fibrin in a vessel (excepting the capillaries) is due to alteration in the composition of the blood, or in the structure

of the walls of the blood vessels. The latter is the most frequent cause of cerebral thrombosis, and is due to atheroma or to syphilitic endarteritis. Acute infectious diseases, exhausting chronic ones (phthisis, cancer, etc.) alter the blood's composition, and may cause thrombosis.

Diagnosis between embolus and thrombosis of a brain artery is largely a matter of age, since an apoplexy in an individual past forty-five and without heart lesion is more likely due to thrombosis than to embolism. Apoplexy in persons under forty and without heart lesion is presumably due to occlusion from syphilitic endarteritis. Embolic occlusion is seen mostly in the young, and is but rarely repeated.

The symptoms of apoplexy from embolism are similar to those from cerebral hæmorrhage, but are less in intensity. Their onset is sudden and without prodromata; those of thrombosis usually have prodromata, sometimes repeatedly during a long period precedent to the apoplectic attack. Embolism affects most often the branches of the middle cerebral artery, and somewhat more frequently the left one than the right; thrombosis has the same predilection, but the arteries of the circle of Willis, the basilar and the internal carotid are not infrequently the site of either form of obstruction, the thrombotic process, however, often extending to neighboring branches not at first affected, thus increasing the gravity of the condition. Syphilitic endarteritis affects particularly the circle of Willis, but

its larger branches, as well as the vessels leading
to it, are often its victims.

The brain tissue dependent upon the blood pre-
viously supplied by a now occluded vessel dies and
undergoes softening, unless there be collateral circu-
lation. This is not possible, except on the cortex,
and then in limited degree, and in the larger ves-
sels leading to the circle of Willis.

Softening has been classified as red, yellow and
white, but the color in red softening is due to the
presence of blood coloring matter, and is found in
parts that are vascular in the normal state (such as
the cortex); by decomposition and absorption of
the blood pigment the red tint changes to yellow,
and then the term yellow softening applies. In the
white matter the softening is nearly white in tint.
The consistency of the softened mass is semi-liquid,
and after its complete absorption its site is repre-
sented by a cicatrix generally, at times by a cyst.

While the symptoms of an apoplexy from vas-
cular occlusion are, in the main, those of a cerebral
hæmorrhage, yet there are differences that will suf-
fice in most instances to determine to which class the
case belongs. In the former the face is generally
not turgid, the respiration less disturbed, the initial
lowering of temperature seen in cases of hæmorrhage
is rarely present, while its rise will not be great
when it does occur. Embolus occluding a brain
artery is more apt to produce convulsive movements
at times epileptiform in character, and, if the vessel

involved be small, there may be no loss of conscious-
ness. In thrombosis, even of a large vessel, there
may be no loss of consciousness, or the latter may
come on only after symptoms of hemiplegia.

The symptoms from occlusion of a cerebral blood
vessel are direct and indirect, and if collateral circu-
lation be established (cortical, internal carotid, etc.),
all the symptoms will disappear. Oppenheim holds
that a thrombus may break down and be washed
away in some cases, and thus explains transitory
hemiplegia. The direct symptoms are those that re-
main, and they are of the same value in localization
as in the case of hæmorrhage.

Aphasia is more common in occluding lesions of
the left hemisphere than in hæmorrhage; hemia-
nopsia and hemianæsthesia are the results if the pos-
terior cerebral is blocked up and no anastomotic cir-
culation is possible (not common).

The prognosis, so far as life is concerned, is more
favorable in occlusion than in hæmorrhage, as a
general rule, but when coma is intense the danger
is rather in its intensity than in its duration (Gowers),
a moderate degree lasting several days being not in-
frequently followed by recovery. Thrombosis of the
basilar artery is almost always fatal, and next in fa-
tality is that of the internal carotid. Danger to life
increases with each succeeding attack of thrombotic
occlusion, and in the thrombotic process there is
great tendency to recurrence; in embolism there is
but little, while in syphilitic vascular disease recur-

rence must be expected if the disease be not ar-
rested.

Inflammatory reaction sets up around the softening
tissue, and it is more severe than the corresponding
process in cases of hæmorrhage, and may give rise to
severe cerebral complications, such as delirium, head-
ache and convulsions, the latter at times a very un-
favorable complication.

If the paralytic symptoms, although not so great
nor so extensive in acute softening as after hæmor-
rhage, do not disappear after two or three weeks
have passed, they will be permanent. From throm-
bosis there is distinct impairment of the mental
faculties; from embolism there is usually but little,
or there may be none at all.

The treatment of apoplexy consists in first placing
the patient in the most advantageous posture. He
should be put to bed and laid on the paralyzed side;
the respiratory muscles of the sound side have thus
the fullest freedom of action and stertor is said to
thereby cease. Cold applications may be made to
the head and warm ones to the extremities, mustard
applications being advised by different authors.

Treatment by drugs at this stage must be almost
wholly symptomatic. The effect of even a small
hæmorrhage within the skull cavity is to set up irri-
tability of the vaso-motor centres, and our remedies if
chosen by the resultant symptoms, will quiet the
vascular excitement. *Belladonna, Glonoine, Melilotus alba*
and *Opium* will be thought of at once. When the

coma is profound, the stertorous breathing marked, the face dusky and the patient covered with sweat, *Opium* is the chief remedy. When the face is red and hot even if coma or stertor be present *Belladonna* should be prescribed; *Glonoine* and *Veratrum viride* resemble *Belladonna* very greatly, but the cardiac irregularity of the first and the more powerful pulse of the second may serve as points of difference. *Melilotus alba* ought to be of service in the early active stage.

The foregoing remedies have been used by the writer in such cases in the third centesimal dilution, excepting *Glonoine* which had better be given in the sixth; in cases of alarming character, the doses may be given every 15 minutes, but the intervals should be lessened as soon as improvement in the pulse or the respiration or the appearance is noted.

When consciousness has been even partly restored, *Arnica radix* should be given in third dilution every two or three hours, a few doses only, after which the symptoms should be studied for special indications and later, when inflammatory reaction has set up, the same method should be followed.

In cerebral thrombosis but little can be done at the beginning, since the occlusion already present will extend, or if not absolute will soon become so. If as a result of the shock vascular symptoms set up, the remedies already mentioned will possibly be indicated, but *Glonoine* or *Baryta muriatica* will at once be thought of. *Lachesis* and *Crotalus horridus* ought to be of value in combatting the reactionary inflammation

and in keeping it within limits. *Phosphorus* and *Sepia* have been highly commended in this stage.

Embolic occlusion, after it has occurred, cannot be remedied. *Aconite* followed by *Arnica radix* 3, ought to be of service in preventing too severe a reaction.

For the hemiplegia or other paralysis following any form of apoplexy, *Causticum*, *Cocculus*, *Nux vomica*, *Graphites*, *Baryta* and *Arnica* are recommended by a number of observers. The writer has seen apparently remarkable results from *Nux vomica*, low, 2x, in hospital cases; and in private practice, *Baryta carbonica* or *muriatica*. It must not be forgotten that the symptoms due to destructive changes in the motor area or motor tract cannot, according to present views, be relieved, and that the other or indirect symptoms can and do disappear without treatment. Nevertheless, the secondary (indirect) symptoms may not disappear unless under the action of suitable remedies.

The use of electricity has been greatly extolled in the treatment of hemiplegia and other paralyses of cerebral origin. As a means of keeping up the nutrition of the muscles of the paralyzed part faradism is of high value, and should the paralysis be only an indirect symptom, its disappearance will then find the muscles ready and able to act.

The employment of static electricity in relieving the secondary contractures in hemiplegia has been much vaunted; the writer has used it repeatedly without the slightest effect in such cases, although it happened that the patients themselves were at first deceived

and believed they had received a greater power and free-
dom of motion in the paralyzed limbs. Hemiplegics,
like other paralytics, learn to use with greater adroit-
ness such trifling power as may exist in the affected
limbs, and thus may honestly believe that a cure is
being effected.

Brain Tumor.

A growth in or upon the brain may irritate or
destroy the parts adjacent to it, or may do both. It
may therefore produce localizing symptoms. It also
in most cases causes general ones which, taken to-
gether, will justify the diagnosis of brain tumor. Such
a mass is only a foreign body, the localizing symptoms
being those depending upon its location, the general
ones being largely due to pressure of the growing
mass. The kind of tumor cannot be known from
either set of symptoms but may often be determined
by other information.

The records of the autopsy table show that certain
parts of the brain are places of election for certain
kinds of tumor. Glioma may affect any part of the
brain but selects first the cerebral hemispheres and
next the cerebellum; tubercle affects mostly the cere-
bellum and next the pons; syphiloma is found oftenest
at the base, next on the cortex in the central and
pre-central regions, while it is rare on the cerebellum
or in the white matter of the cerebrum.

The existence of morbid growths elsewhere in the

body would make it probable that a tumor of the brain is of the same nature.

Age is at times of great help; a brain tumor in a child is evidence that it is probably tubercular in character, while a tumor in a person of advanced years is more probably glioma (including sarcoma) than syphiloma, the reverse being the case in the young adult.

The rate of progress in growth (as shown by increasing intensity of the symptoms) may be of corroborative value. Glioma grows slowly and being itself liable to hæmorrhages may give rise to apoplectic attacks; carcinoma grows rapidly but it is usually metastatic in origin, while syphiloma grows rapidly in one direction and at the same time breaks down in another, accounting thus for the changeability of its symptoms.

The general symptoms of brain tumor usually precede the appearance of the local ones. Headache exists in the great majority of cases; it is mostly continuous, but has spells of aggravation. Its intensity is often frightful, and its character varies in different cases; it is aggravated by forced exertion, stooping, coughing, etc. Its subjective location may be frontal, occipital, lateral or diffusely spread, but the subjective location is of no value in localizing the site of the tumor; the head is often sensitive to percussion with the fingers and at times extremely so in the neighborhood of the growth.

Choked disk is considered by Oppenheim to be

characteristic of the existence of tumor, nine out of every ten cases of it being so caused. He differentiates the swollen choked disk from a simple optic neuritis, which also can exist as a result of brain tumor, but which is to be seen as well in other diseases. It has been observed that the vision, during the course of brain tumor, does not fail to the degree expected with such swelling and obliteration of the disk; the vision does finally disappear if the patient live for a considerable time, the ophthalmoscope then usually revealing consecutive atrophy of the nerve.

The mental state suffers generally during the course of a brain tumor, varying from slight loss of power of attention or concentration to an absolutely stupid condition, or apparently as if overcome by an opiate. Conditions of insanity develop quite often, mostly of the type of mania, occurring in paroxysms, or at times of hallucinatory insanity or melancholia, but more often the mental change is evidenced by silly, foolish behavior —in one case the writer has seen marked hysterical manifestations. Growths at the base or distant from the frontal lobes, and tumors of small size, are not so apt to cause psychical change.

Vertigo is frequently an accompaniment of brain tumor; if violent it becomes a localizing symptom. It is not attended by nausea, generally. Vomiting, projectile in character, is noted in the majority of cases; if associated with headache, as it often is, it may give rise to the erroneous diagnosis of migraine.

Slowness of the pulse is observed when intra-cranial

pressure becomes considerable; it is not an early symptom unless the structures beneath the floor of the fourth ventricle become implicated, and then it has a localizing value.

Attacks of convulsions with or without loss of consciousness, or of the latter alone, or of temporary blindness, are among the less frequent general manifestations.

Localizing symptoms vary with the site of the tumor; they may be irritative in character, or paralyzing, most often the latter. The symptoms are often indirectly produced, especially when intra-cranial pressure is great.

Tumors of the frontal lobes are apt to cause marked mental changes already mentioned; if left-sided, motor aphasia will be present when the motor speech area becomes affected; if right-sided there may be no localizing symptoms.

Tumors involving the central convolutions are apt to cause motor irritative symptoms in the beginning, such as spasm in one definite part of the body; the spasm is repeated at varying intervals, and in time paralysis of the part (monoplegia of face or arm or leg) comes on. General convulsions may result, epileptic in character, but beginning in the same definite part (Jacksonian epilepsy). With the growth of the tumor the paralysis spreads until a hemiplegia is produced, with strong tendency to contractures. In most cases there is some hypæsthesia or paræsthesia in the paralyzed side.

Tumors affecting the cortex behind the motor areas must interfere with the muscular sense, and, if in the neighborhood of the supramarginal and angular gyri of left hemisphere, will cause alexia.

Tumors in the occipital lobe, if involving the cortex adjoining the calcarine fissure, will produce homonymous hemianopsia; if affecting the outer face of the occipital lobe, especially the angular gyrus, there may be loss of the memories of the uses of things previously well known, such as the objects seen in everyday life; this condition is known as psychical blindness. If the tumor involve the anterior area, it may encroach on the angular and supramarginal gyri, with the result of causing alexia, if on the left side. Hemianopsia is also caused when the tumor affects the white matter of the occipital lobe, since the optic radiations will be affected.

Tumors of the temporal lobe, if left-sided and involving the auditory speech area, cause word-deafness with paraphasia; otherwise such tumors of either side cause no direct symptoms.

Tumors of the centrum semiovale are not specially characterized by their symptoms, but the nearer they are to the cortex the more will cortical symptoms appear; hemiplegia of gradual onset is considered to be symptomatic, while, if the corpus callosum be specially implicated, extension of the growth will probably cause paraplegia. Tumors of the corpus callosum are also believed to cause a marked stupidity, apathy or soporous condition. (See Fig. 45, page 261.)

Tumor at the base can hardly help affecting some of the cranial nerves. If in the anterior fossa it is likely to affect by pressure the olfactory and optic nerves and, by extension of pressure, the third nerve and first branch of the fifth. If upon the sella turcica, the optic chiasm, the hypophysis, the nerves of the orbit and the first branch of the fifth; if in the lateral cavity of the middle fossa, all branches of the fifth may be affected and also the ocular nerves.

Tumors of the posterior fossa will by pressure affect the nerves arising from the pons and medulla oblongata, directly or indirectly, except the twelfth nerve, which by its low point of emergence may escape direct pressure.

Tumor affecting one crus cerebri will usually cause hemiplegia of the opposite side, with third nerve paralysis of the same side. (See Fig. 38, page 236.)

Tumor in the corpora quadrigemina and the structures just below it, causes ataxia and paralyses of the eyeball muscles; the ataxia is of the cerebellar variety.

Tumors of the pons and medulla are infrequent. If limited to one side of the pons, a tumor may cause hemiplegia of the opposite side of the body, when it is above the middle; if below the middle, it will probably cause alternating hemiplegia, that is, paralysis of arm and leg of opposite side with paralysis of the face on same side, and the latter may show degenerative reaction. If the tumor is low down in the pons, the face escapes. Tumor in the medulla or

pons, involving the neighborhood of the sixth nerve
nucleus, will cause paralysis of that nerve, and there
is then often conjugate deviation of the eyes to the
other side. (See Fig. 38, page 236.)

Tumor in the medulla, if on one side, will cause
incomplete hemiplegia, arm and leg of opposite side
being affected, but with paralysis of the tongue on
the side of the lesion. If the growth involve both
halves of the medulla, the symptoms will be those
of bulbar paralysis with, possibly, paralysis of the
four limbs. If it invade the fibres forming the in-
ferior peduncle (medulla), or those of the middle
peduncle (pons) of the cerebellum, ataxia, cerebellar
in character, is to be expected.

The direct symptoms of tumor of the cerebellum are
occipital headache, extending at times to the nape and
even lower, vertigo, and cerebellar ataxia. The
ataxia disappears when the patient is lying down;
when up and walking about it is a reeling gait, or
if slight, it is as if he were in fear of falling; the
ataxia does not affect the arms. The forced move-
ments seen in cerebellar troubles, turning continually
to one side when lying, or going to one side when
walking, cannot as yet be positively ascribed to disease
of any definite part of the cerebellum, although both are
cerebellar symptoms; they have been noticed in con-
nection with involvement of the middle peduncle of
the cerebellum. Tumors of the middle lobe of the
cerebellum, if growing downward, will by pressing
upon the floor of the fourth ventricle give rise to

either irritative or paralyzing conditions in the nerve nuclei and nerve centres below.

The connections of the cerebellum with other parts of the brain and cord are large peduncles; a tumor may, by pressure on one, draw upon and possibly displace some other structure, and so give rise to confusing indirect symptoms; the effect of a tumor anywhere in the brain is at times greater in this displacing result than by its direct action in its own site.

Vomiting is a symptom of cerebellar tumor very frequently seen, but is not necessarily a direct one. Nystagmus is at times a symptom of cerebellar tumor, due probably to distant effect on the quadrigeminal region; internal hydrocephalus is at times observed, a direct result of pressure on the vena magna Galeni, with consequent damming back of the circulation.

Diagnosis. Some of the general symptoms of brain tumor occur with other disease processes. Severe optic neuritis may occur with nephritis, with meningitis, and even with hydrocephalus, while headache can exist in all of these; the microscope, the history, progress and special symptoms will serve for differentiation. Abscess of the brain rarely causes choked disk, and generally there is an etiological factor present (ear-disease, suppuration elsewhere, injury to the head). Poisoning by lead may cause choked disk, headache, delirium and convulsions, but these are preceded by other evidences of the poison.

Profound anæmia may cause headache, and even choked disk; here the existence of the cause, as well as its relief by treatment, will clear up any doubt.

Chronic alcoholism may cause optic neuritis, head-ache, etc., but the optic affection is characterized by central scotoma for colors and the alcoholic tremor is present. Hysteria at times presents a functional hemi-plegia, with headache that might lead to doubt; the headache is relieved by emotional influences, yet it is not rare for hysterical symptoms of marked type to appear during the progress of brain tumor. Syph-ilitic meningitis is especially apt to appear at the base and give rise to the symptoms produced by a tumor in that situation.

The operative treatment of brain tumor has been the subject of great attention. The indications for it have become sufficiently definite to be tabulated through the experience of v. Bergmann, Horsley, and others.

The tumor must be susceptible of accurate localiza-tion; its location must be accessible to the surgeon; it must be near the surface; it must be limited in out-line, or at least not diffuse or infiltrating in its mode of growth. The surgeon is thus limited practically to tumors in or upon the motor area and the lower parietal area.

The earlier an operation can be done, the better. In cases not suitable for operation for removal of the growth, trephining with opening of the dura will

relieve brain pressure, and so relieve many of the symptoms.

Intracranial aneurism of a large vessel acts upon the nervous structures by pressure, and is to all intents and purposes a brain tumor. The exciting causes are a previous embolus of infectious character, which sets up inflammation in the vessel's wall with subsequent yielding; syphilitic disease of the arteries; primary arterio-degeneration in later life and (rarely) injury to the head.

The arteries of the brain most frequently affected by aneurism are the middle cerebral, basilar and internal carotid. In two-thirds of the cases of aneurism one of these three vessels is the seat of the disease (Gowers). Much less frequently affected are the anterior cerebral, posterior communicating, anterior communicating, vertebral, posterior cerebral and inferior cerebellar — the last two very rarely.

The diagnosis is extremely difficult, and is often impossible. In rare cases it becomes certain when a *bruit* can be heard on auscultating the skull. The symptoms being those of a tumor at the base, aneurism may be suspected if a lesion can be located in the neighborhood of the great vessels and the patient have general arterial disease or organic cardiac affection.

The prognosis, when the diagnosis of aneurism has been made, becomes very gloomy, since rupture may occur at any time; rupture does not neces-

sarily occur, and when it does, is not necessarily fatal.

The treatment of tumor of the brain by homœopathy has been considered at length by the writer in Hahnemannian Monthly, 1896. As the symptoms are those due to pressure upon and irritation of different parts of the brain by the growth, the latter is practically the same as a foreign body, and if possible, it should, as in the latter case, be removed mechanically. To administer remedies selected according to the symptoms caused by the pressure upon and irritation of the invaded structures is only to help those structures tolerate better such interference. The time may come when by homœopathic medication morbid growths may be made to retrogress; until that time, brain tumor must be considered as not the subject of homœopathic treatment. Palliative and antipathic measures will have to be employed; potassium iodide in ordinary doses in some cases has given considerable relief for a time. Trephining the skull has been resorted to for relief of intracranial pressure and with success, in one case even to retrogression of optic neuritis.

Intracranial aneurism will be best treated by the use of *Baryta muriatica* and *Plumbum* in persons not syphilitic. The general symptoms may call for any other drug and such should be given. In cases having a syphilitic history, *Mercurius protoiodatus* 2x, or if much mercury have already been taken,

Aurum muriaticum natronatum 6x may be of value.
In any case the writer would give in addition a
nightly dose of *Syphilinum* 200 or higher for a
month, repeating the prescription after an interval
of a week or two.

Brain Syphilis. The effects of the malign influence
of the microbe of syphilis (or of its toxine) within
the skull cavity, are chiefly upon the meninges and
the walls of the blood vessels. The most common
manifestation is chronic gummatous meningitis situated
at the base and especially in the intercrural space and
extending over the chiasm. The process results in
an exudation of gummatous material, alteration of the
blood vessels and injury, partly by compression, partly
by invasion, of the nerves. The symptoms are there-
fore those of a basal meningitis with special implica-
tion of the optic nerve and the motor nerves to the
eyeball. Other nerves are at times affected.

With the inflammatory changes mentioned the ar-
teries are also involved, the walls being thickened
and their lumina narrowed, often resulting in throm-
botic occlusion.

Next in frequency is gummatous meningitis of the
convexity, involving more or less the cortex and then
giving rise to wide-spread softening.

The gummy exudation may be in masses of vary-
ing size and thus act as tumors, but the tendency to
fatty degeneration and caseation is very great and
leads to a changeability in the symptoms at times
surprisingly rapid. Implication of the middle of the

chiasm will cause bi-temporal hemianopsia, but vary-
ing changes in the visual field point to the syphilitic
character of the affection. Similar instances are the
receding of a third nerve paralysis until it has dis-
appeared, perhaps to recur in a few days.

The intra-arterial proliferation is characterized by
great permanency, and occlusion of the proper artery
will cause a typical hemiplegia.

It is not infrequently preceded by several light
apoplectic attacks, with hemiplegia lasting from a few
minutes to a few days.

Endarteritis of the basilar or vertebral artery can
give rise to bulbar symptoms, but such symptoms may
occur if an exudation extends as far backward as the
pons and medulla.

The general symptoms of chronic gummatous me-
ningitis are headache, vertigo, vomiting, attacks of
mental confusion or of mental incapacity ; in some
cases excitement. Polydipsia and polyuria are often
present. The headache is at times of great intensity
and is often, but not always, worse at night. Its
cessation may deceive the unwary physician into be-
lieving that he has relieved it, but it frequently ceases
just before a paralysis occurs, such as hemiplegia,
paralysis of eye muscles, etc.

Optic neuritis or even choked disk occurs in more
than two-thirds of the cases, but not always on both
sides.

The diagnosis of brain syphilis may often be made
without any history, by noting the changeability of

the symptoms, their coming and going, their multiplicity. When, in addition to these points, evidences of a previous general adenitis, (post-cervical, epitrochlear or inguinal glands) or atrophic scars on face, legs, etc., if present, relieve us of the necessity of questioning too closely an unwilling patient. A married woman with no suspicion of the cause of her head symptoms may be questioned about early miscarriages, still-born children, infant born with eruption around the anus and suffering from snuffles, etc.; her affirmative answers will tell the story.

The treatment of brain syphilis has been given under the head of brain tumor.

Cerebral Infantile Paralyses. Spastic Infantile Paraplegia. The cerebral paralyses of children are most often determined by disease or injury to the brain during the first few years of extra-uterine life, next in frequency during parturition, and least during intra-uterine life.

Of the first, the greater number occur during the course of the acute infectious diseases or possibly by an infection *sui generis*, or as the result of traumatism upon the head.

The second includes those where the act of parturition is difficult and prolonged, especially when the forceps have been used, not because the instrument was applied but because of the conditions demanding it. The result of the great pressure is meningeal hæmorrhage usually over the upper parts of both motor areas, or direct injury to both these areas by

pressure of the yielding parietal bones. The conse-
quence is then not hemiplegia but paraplegia.

The third kind, beginning during intra-uterine life,
has been attributed to different causes, chiefly to in-
jury to the mother during pregnancy (especially injury
affecting the uterus), occasionally to severe emotional
shock, and to disease affecting the fœtal brain and
resulting in faulty development, particularly of the
central convolutions.

The symptoms of the first class occur in the majority
of cases during the first few years of childhood as
an acute attack of fever accompanied by headache,
vomiting and general convulsions; in some cases the
convulsions may be confined to one side; the con-
vulsions may be repeated or may be absent. The
symptoms of the acute stage may continue a day or
two, or may last a week or more. When this stage
has passed it is noticed that the child is paralyzed,
the symptoms being those of hemiplegia with but tem-
porary implication of the face; in children who can
talk there may be temporary aphasia. Sensibility is
usually not disturbed. In the course of time some
improvement is manifested in the paralyzed limbs,
but the rate of growth is less rapid than in those
of the sound side. Meanwhile, contractures of the
flexors and adductors come on which are, in the arm
like those of an ordinary hemiplegia, but in the leg
the typical hemiplegic gait is absent, because there
is some flexion at the knee. Or, certain spasmodic
movements, athetosis or chorea, appear in the affected

arm and hand especially, or associated movements
occur in the affected side when movement is attempted
by the sound side.   The knee-jerk is usually exagger-
ated and ankle clonus can generally be elicited.

In a large number of the cases epilepsy develops
in course of time, occasionally continuing from the
termination of the acute stage.

The mental development is nearly always affected
in some degree, from mere slowness of perception
to imbecility or even idiocy.

The second class, arising from injury during birth,
generally show on coming into the world evidences
of severe pressure upon the head, and cyanosis, while
the history tells us that the accoucheur had difficulty
in bringing the infant "to life."   Convulsions may be
present at this time but often cease, to return some
weeks or months later.   The child does not develop
as it should; it is found that it does not use the
limbs of either side and that both trunk and neck
muscles are affected, since it cannot hold the head
up nor sit if unsupported.   This condition is a double
hemiplegia and indicates extensive injury to both motor
areas.   When, as frequently happens, the upper por-
tions only of the motor areas have been involved,
the effect is seen only in the lower extremities and
the condition is a spastic cerebral paraplegia.   Some
rigidity and contractures appear.   When attempts are
made to teach the child to walk it refuses to put its
feet to the floor, indeed draws them up and when it
does learn to take some steps (by assistance) the ad-

ductor muscles of the thighs are seen to be contracted, sometimes to the extent of crossing the legs. Should this condition remain, the child when able to walk does so by "cross-legged progression." Mental defect is common in this form and epilepsy may be expected to develop.

The third class of cases, those due to intra-uterine causes, are usually double hemiplegias having the symptoms of the first class without the acute stage, fever, etc. The mobile spasms, or choreic movements, are the same, but the mental impairment is, of course, much greater. Epilepsy develops in a majority of the cases.

The differentiation of the three types of cerebral palsy in children is partly a matter of the history, partly one of examination.

A child having been perfectly well up to, say, the third year, without history of injury already described at the time of birth, and without any evidence of faulty development *in utero* (such as abnormalities in the shape or size of the skull, gothic palate, etc.), is then attacked by an acute affection following the course and having the sequelæ already stated. The only conclusion to be drawn is that the affection is acute encephalitis. When evidences of faulty intra-uterine development are present, the case belongs in all probability to the third class, though by no means all of this class show such evidence.

The history of difficult labor, the child being asphyxiated when born, will classify the remaining cases;

the history of the condition at birth cannot always be obtained.

The lesions in the cases that are intra-uterine in origin, or which are caused at the time of birth, result in agenesis of the motor (and other in some instances) cortical areas, and agenesis of the motor tracts. The motor convolutions do not develop and their shrunken remains are at the bottom of a narrow fossa at the site of the Rolandic fissure and may open into the lateral ventricle. This condition is known as porencephaly. The gap may be filled by a cyst.

In the hemiplegic cases, porencephaly of one hemisphere may exist or partial atrophy and shrinking of the central convolutions, with consequent degeneration of the pyramidal tract; at times the cortical areas show but little gross change and these are the cases in which epilepsy develops later with little or no motor disturbance.

It has been already stated that in these forms of brain trouble the mentality is more or less affected. The child may be absolutely idiotic, may be imbecile, or may be only backward, the condition not being observable until its deficiency is remarked when compared with the average child of the same age. Many do not learn to talk until very late; some never learn. Epilepsy develops in about half of the first form and in more than half of the others.

Cruelty to others and destructiveness in general are often seen when the mental development is low. In some cases the children have some special habit of

self-injury, as beating the head with the closed fist, or knocking it against the floor, so that the tissues of forehead or occiput are kept constantly bruised.

The diagnosis of cerebral palsy in early infancy is not difficult; the increased reflexes and absence of reaction of degeneration distinguish it from paralysis following poliomyelitis, with which it might possibly be confounded.

Prognosis. In the first form, if the mental development has not suffered beyond a slight degree, the hemiplegia gradually lessens or may practically disappear, and the individual may attain adult age with, it may be, some peculiarity of gait or some asymmetry of the limbs of the two sides. In the second and third forms, the injury to the brain is relatively great, and when mental development is poor and especially if spasms occur, the prognosis for life beyond the period of puberty is not good.

Treatment of the cerebral palsies of children must be educational, hygienic and medicinal. If the child is unable to talk, the writer recommends that it be placed in some hospital for children, and believes that from this association with many children the latent cortical areas for speech will be aroused into activity sooner than through teaching by adults.

When the child has practically no power over its limbs and yet its mental state is not injured to a very great degree, the writer employs faradism to the unused members, causing contractions of the muscles of the extensor side, and acting upon single muscles.

Not only does this stimulate the growth of the muscles, but it also, he believes, teaches the child's brain how to act upon them.

Cases that are imbecile or worse, need the care of an attendant and cannot be taken in a hospital. They should be sent to some institution for the care of feeble-minded children. To care for such cases at home is a trying task to the parents, unless they are able to keep a special attendant for the child. The home care of such a patient is apt to develop many of the emotional defects so often seen in later years.

Surgical interference may be advisable in cases of old contracture, and braces and similar appliances may enable the child to walk. In such matters, each case must be taken by itself and treated accordingly.

The remedies applicable in cases of cerebral infantile paralysis will vary according to the associated conditions of spasm, feeble mental development, mutism, etc. The writer has used *Baryta carb.* and *Natrum mur.* in the hope of developing such parts of the cortex as were not wholly beyond such possibility. Other remedies were used from time to time, according to special indications, among these *Sulphur*, *Calcarea carb.*, *Psorinum* and *Silicea* being preferred. In cases without spasms, many relative cures have thus been made, and some absolute ones, but it is impossible to say how much was done by the educational influences already mentioned, and how much by the remedies administered.

The epileptic cases have in some instances been

greatly improved, *Belladonna, Hyoscyamus, Cicuta virosa,*
Cuprum aceticum and *Cypripedium pubescens* 3, having
been the chief remedies employed; the latter has
been found of especial value in lessening the irrita-
bility of the cortex in cases having many minor seiz-
ures, apparently provoked by some trifling error in
diet, a slight cold, etc.

Acromegaly. As the name implies, the symptoms
of acromegaly are most noticeable as enlargements
of the ends of the body, the hands and feet, especially
the fingers and toes, but also the nose, lower jaw,
lower lip and tongue. Besides these, the clavicles, the
sternum, and the upper vertebræ show enlargement
and deformity, the latter being a kyphosis or kypho-
scoliosis of the cervico-dorsal region.

The disease begins in early adult life, usually be-
tween the ages of 20 and 30, with paræsthesias or
slight rheumatic pains in hands and feet. Gradually
the hands and feet enlarge, as well as the parts
above mentioned. The enlargement appears to affect
all the structures, but especially the bones, which
become thickened rather than elongated.

The patients become apathetic, sleepy, at times
stupid, and complain of great weakness. In women
the menses cease early in the disease, and the uterus
is often atrophied. In men there is weakness or loss
of the sexual power.

The chest becomes deepened, with kyphosis at the
upper vertebræ; there may be an associated and
compensatory lordosis in the dorso-lumbar region.

Hemianopsia, most often bi-temporal, has been repeatedly observed, and occasionally optic neuritis.

Heredity plays no part in the disease; trauma has been given as the exciting cause; inhalation of illuminating gas (to unconsciousness) was followed in one case by the appearance of the disease, and it has been stated that several cases have followed removal of the ovaries. The post-mortem findings have been various. Persistence of the thymus gland, enlargement or smallness of the thyroid, have been noted, but far more often, growths in the vicinity of the hypophysis cerebri, or tumor or degeneration of that body, so often, indeed, that the pituitary gland is now considered to be, when diseased, the cause of acromegaly. The function of the gland being unknown, a theory of its influence in causing acromegaly cannot as yet be made, but hemianopsia, optic neuritis, and some of the cerebral symptoms may be explained as the result of pressure on the chiasm or optic tracts, the crura cerebri, or even on the carotids.

The course of the disease is slowly progressive, but in many cases after reaching a certain height it ceases to advance.

The diagnosis is to be made from the condition of giant growth, which is an anomaly of development and is without the disproportionate enlargement of hands, feet and chin (such cases seem to have a disposition to general dystrophies, particularly acromegaly. M. Sternberg, Zeitschr. f. klin. Med. Bd. XXVII.); from a very similarly appearing disease described by Marie

and termed hypertrophic pulmonary osteo-arthropathy, in which the presence of a pulmonary or pleural affection, the knobbed ends of the distal phalanges of fingers and toes, with fragile and deformed nails, serve for differentiation.

Homœopathic treatment must be guided by the totality of the symptoms. In a case of the writer's, reported in N. A. J. of Homœopathy, 1888, improvement in the general symptoms (mental and physical hebetude, etc.) followed the use of *Sulphur* and *Silicea.*

The Cerebellum

Consists of two hemispheres and a middle lobe known as the vermis. Its external layer is gray matter arranged in more or less parallel narrow convolutions; the fissures extending deeply, and branching by shallow sulci give a foliated appearance to a section of a hemisphere. On both the upper and lower aspects of the hemisphere distinct lobes are evident. The gray matter of the cerebellar hemisphere is in relatively greater amount than in the cerebrum.

The middle lobe or vermis is divisible into a number of parts; the sulci are deep, but give off shallow branchings. The gray matter of the vermis is relatively small in amount.

Within the white matter of each hemisphere, near its junction with the middle lobe, is a hollow gray body similar in appearance on cross section to that

of the olivary body in the medulla oblongata. It is termed the corpus dentatum. Near the latter, towards the middle lobe, is a small nuclear mass, the emboliform nucleus, and still further towards the median line another, the globose nucleus.

The gray cortex of the cerebellum is made up of three layers, an outer or molecular, and an inner or granular one, while between them is the layer of large cells, the cells of Purkinje. Their dendrites are noted for the immense number of branchings directed outward, while the neuraxones pass inward.

Practically very little is known of the functions of the cerebellum. Its connections with the cerebral cortex *via* interpolated gray nuclear masses in the pons (middle peduncle); with the spinal cord *via* the restiform body (inferior peduncle); and with the subthalamic region *via* the red nucleus of the tegmentum (superior peduncle), show its functions to be various and important. All, however, seem to be centred in the middle lobe, for removal of a cerebellar hemisphere is not followed by any characteristic symptoms.

Disease of the middle lobe (or indirect action upon it) causes cerebellar ataxia, attacks of vertigo (especially the feeling as if being rapidly rotated), and at times a disturbance of speech like the "scanning" speech.

Slowly destructive lesion of the middle peduncle can progress without giving rise to symptoms. Irritative lesion often causes compulsory movement of

the body — in walking, going to one side; in lying, turning on the long axis of the body.

Nystagmus has been repeatedly noted in the course of cerebellar disease (the superior peduncle has direct relation to the red nucleus which is in the neighborhood of the rootlets of the third nerve). Symptoms from pressure or irritation of neighboring structures occur at times in the course of cerebellar disease, especially the cranial nerves, whose nuclei are in the pons and medulla. The corpora quadrigemina may be thus affected and bilateral ophthalmoplegia (exterior eyeball muscles only) appear, preceded by cerebellar ataxia. If the ataxia follow ophthalmoplegia the lesion is of the corpora quadrigemina.

Choked disk is a very frequent accompaniment of cerebellar tumor.

Hemiplegia may be caused by cerebellar tumor; it may be on the side of the lesion or the reverse, according as the site of the growth is below or above the decussation of the pyramids.

Treatment of cerebellar disease will be found under Brain Tumor and Abscess of the Brain.

Headache.—Cephalalgia.

Headache is a very common affection and may proceed from the most diverse causes. It is well known as one of the manifestations of cerebral congestion, meningitis, brain tumor, etc., of the infectious diseases, of poisoning by alcohol, lead, nicotine, etc., of anæmia, of aortic valvular disease, of nephritis, of

constipation, etc. It may be caused reflexly by in-
digestion, intestinal disease, uterine disease, by dis-
ease of the naso-pharynx, catarrh of the frontal sin-
uses, disease of the middle ear, uncorrected errors of
refraction and faulty balance of the eye-ball muscles.

Besides these and other symptomatic and reflex
headaches in some, no such cause can be assigned or
at least traced; they are apparently a constitutional
neurosis, since heredity often has a part in their ex-
istence.

The diagnosis of the different forms of headache
requires a thorough examination in all directions in
order to ascertain the cause. *Tolle causam* must be
the watchword when undertaking the treatment of
the disorder, and when possible to follow the injunc-
tion the success resulting is in the highest degree
gratifying.

Headache is located within the skull cavity and
must be distinguished from neuralgic or rheumatic
pains in the tissues external to the bones; frequently the
two conditions are combined. The ache is generally
diffuse, but often is concentrated or limited to certain
regions; predominant are frontal, temporal, occipital
and vertex regions as locations of headache, and at-
tempts have been made to connect the location of
the ache with some special causal condition acting
either directly (toxic) or reflexly.

The treatment of headache must include first the
removal of its cause by mechanical, surgical and hy-
gienic methods, the correction of errors of refraction

or other ocular troubles, etc.; next, the selection of
a remedy based upon all the symptoms of the patient,
especial prominence being given to the conditions of
aggravation and amelioration, the character of the pain,
its site, etc. To give here all the remedies that have
caused headache in the provers would require many
pages, while a mere repetition of the names of those that
have been frequently beneficial in the condition would
be of no practical value. In Lee's Repertory of Char-
acteristic Symptoms under the rubric, headache, un-
defined, 230 remedies are given; while the sub-rubrics
of the different modalities, location of pain, etc., have
considerably more than 200, some of them with but
one remedy, others 50 or more.

To write down all the peculiarities of the headache
and to take time to study out the remedy, will give
far better results than to prescribe off-hand from one's
memory of the *materia medica.*

When a remedy thus studied out relieves one attack
and fails in the second or third, the writer takes this to
be evidence of some distant cause. Even in such case
our remedies will in the long run do better for the
patient than the antipathic or palliative treatment by the
coal-tar products, or the older sedatives and hypnotics.
Thus a case of recurrent headache during many years
was examined at different times for a possible cause.
Eyes, ears, nasal passages, pelvic organs, etc., were
thoroughly examined without finding anything whose
correction relieved. The peculiarity of the headache
was an accompanying dull ache in the abdomen. On

this peculiarity *Conium maculatum* was prescribed and with almost immediate relief.

A second time it relieved, but afterwards it failed utterly. Then, *Cinnabar* was given with great benefit at first, but later it was useless. In Lee's Repertory 38 remedies are given for the combined abdominal and cephalic pain; most of them had other symptoms contraindicating their employment in the case. For several years the patient has been greatly benefited by the use of *Dioscorea* (one of the 38), the attacks being generally lightened and frequently cut short— the dose is a few No. 10 pellets saturated with the tincture, repeated every few hours until some relief is felt. In the same case acetanilide, antipyrine and phenacetine have been used with either no result at all, or if improvement occurred it lasted only as long as the drug was present in the system, the head-ache returning the next day.

Vertigo.

Vertigo is the sensation of instability in the sub-ject, or the false appearance of motion of external objects, or both. It is a symptom arising from many causes and is found in organic disease of the brain, especially tumor of the cerebellum, in cerebral hyperæmia, anæmia, arterio-sclerosis; in altered com-position of the blood by toxic influences occurring in dyspepsia, constipation, and uræmia; by poisons in-troduced from without, the chief ones in every-day life being alcohol and nicotine; with disease of the

inner ear (semi-circular canals), occlusion of the Eustachian tube, even a mass of cerumen pressing on the tympanic membrane; from the faulty projection of objects in paralysis of eye-ball muscles.

Vertigo may be a symptom of neurasthenia and it may be the equivalent of an epileptic spasm.

The attack comes suddenly, lasts a very short while; the sensation is as if the sufferer were whirling or as if the ground were waving, or rising, or falling. In many cases the patient feels the contents of the cranial cavity to be whirling or "seething". The attack is often accompanied by faintness, mental confusion or even syncope; nausea and vomiting are frequently observed.

In a person subject to vertigo, an attack may be brought on by a sudden change from the lying to the sitting posture, by turning around suddenly or by stooping.

In old persons with arterial changes vertigo may be a forerunner of apoplexy.

The treatment of vertigo must be that of its underlying cause when such can be discovered. In many cases the cause can only be assumed, but in any case the remedy is to be selected only after study of all the conditions and concomitants. In the Symptom Register to the Encyclopedia more than 400 drugs have vertigo as a symptom in the proving. In Lippe's Repertory of Characteristic Symptoms 103 remedies are given under Vertigo, and of these more than one-fourth are in italics. The sub-rubrics under the

general heading in both the Symptom Register and
Lippe's Repertory, facilitate greatly the choice of the
remedy.

The peculiarity of the vertigo itself will at times
enable us to select a curative remedy ; thus, a vertigo
that was one-sided, so the patient asserted, was entirely
relieved by *Conium mac.* 3, and the feeling that the floor
or ground was wavering under her was cured by
Teplitz spring water, potentized to the third or fourth.

Tropho-Neuroses.

Progressive Muscular Dystrophy Under this term are included all cases of progressive muscular atrophy that are not of spinal origin.

As the affection is a "family" disease, appearing in several members of the same generation and in those of successive generations, it is considered to depend upon some fault in the germ, in the embryonic plan of the muscular system.

Although the trouble is seen much more frequently in males than in females, yet the line of descent is usually through the female.

There are four chief types of the affection, each characterized by symmetrical selection of different sets of muscles, and each first appearing at one definite period of life. Yet members of the same generation may exhibit the disease in different types, and transitional forms of the affection exist—all showing that it is, whatever the type, but one and the same disorder.

The anatomical changes found in the disease are limited to the muscles, the constituent fibres of which are hypertrophied at first, and later atrophy. In the latter period the atrophied fibres are seen thinned, with fissures and vacuoles in their interior, or in fragments with great increase of their nuclei. Connective tissue proliferation replaces the destroyed fibres, or is found between them, or fat deposits occur, both

conditions at times giving the appearance of hyper-
trophy of the part. Pseudo-hypertrophy is absent in
most of the types of the affection.

The chief types of the disorder are: 1, the pseudo-
hypertrophic, beginning between the ages of four and
nine; 2, the infantile form, with affection of the
shoulder girdle as well as implication of the facial
muscles, beginning below the age of four; 3, the
juvenile form, affecting the muscles of the shoulder
girdle and beginning in youth or later.

A so-called hereditary form is like the first form,
but without pseudo-hypertrophy; it begins between
the eighth and tenth years.

Pseudo-hypertrophic paralysis begins, as stated,
in childhood. The parents notice that the child falls
easily, has an awkward, waddling gait, and cannot
stand erect. This excites remark, as the child gener-
ally has apparently well-developed or over-developed
calves and thighs. When standing there is marked
lordosis of the spine from weakness of the glutæi
muscles; at the same time the abdomen is protruded.
The lordosis disappears on sitting and a reverse curve
is then apparent (weakness of the erectors of the
spine). Going up stairs is difficult without the aid
of the arm pulling on the banister; he cannot rise
if lying, except by getting on all fours and then
throwing one hand quickly to grasp the thigh, follow-
ing this action by a similar one with the other hand
— thus climbing up the thighs (Gowers)— all due to
weakness of the glutæi muscles.

In course of time the muscles about the shoulder become affected, usually without pseudo-hypertrophy, often with abnormal position of the scapula. Occasionally a muscle will show a real hypertrophy. The face is involved only exceptionally.

There are no sensory disturbances; the skin over the affected parts is often cold and of bluish mottled appearance. The tendon reflexes disappear when atrophy has advanced in the related muscles. There is no involvement of the bladder.

Contractures may occur in the affected muscles, especially the gastrocnemii, with consequent talipes equinus.

The presence of imbecility, epilepsy or other cerebral disturbance must be considered as a complication.

The **infantile type of muscular dystrophy** is distinguished by wasting of the facial muscles proper, the masseters not being affected. The orbicularis of the eye and that of the mouth waste early. In consequence the eyes cannot be completely closed and the lips protrude somewhat as a snout. Movements of the lips in closing the mouth, in smiling, whistling and speaking are greatly impeded.

When the other muscles of the face are affected, emotional expression is gone, and the face assumes a mask-like appearance.

The process next extends to the muscles of the shoulder girdle, and still later to those of the pelvic girdle. The facial atrophy may precede or follow that of the shoulder or pelvic region.

The **juvenile form of muscular dystrophy** (Erb's form) appears first in the muscles of the shoulder girdle, as follows: the pectorals, trapezius, latissimus, serratus magnus, rhomboideus, biceps, the flexors of the forearm and the triceps. The sterno-cleido-mastoid, levator anguli scapulæ and deltoid escape for a long time, and the forearm and hand muscles are but rarely involved. The wing-like position of the scapulæ is well marked in this form.

Later, the muscles of the pelvic girdle become affected and those of the thighs and legs, the calf muscles usually escaping; then the functional symptoms are like those seen in the pseudo-hypertrophic form.

Pseudo- and real hypertrophy may be observed in single muscles.

Types of progressive muscular dystrophy cannot at times be made out according to the descriptions just given; indeed, from the latter it will be seen that the longer the patient lives the greater the tendency to resemblance of cases among themselves.

The progress of the affection is slow, yet this statement needs qualification. The earlier it appears and the greater the pseudo-hypertrophy, the more rapid will be its course. Gowers maintains that it is almost certain that any child with pseudo-hypertrophic paralysis will not live to reach adult life.

In the other forms the duration of the disease is from ten to fifty years. Its progress is rarely at a uniform rate; it may cease to advance for long periods (24 years in one of Gowers' cases), or, after

a cessation lasting for years, it may affect a new part and in a very short time a third.

The electro-irritability of the affected muscles is simply lessened in proportion to the amount of atrophy; RD is not present. Fibrillation of the affected muscles is rare.

Prognosis.   There is no hope of cure, but the long periods of cessation of the disease that have occurred in many cases give some hope that the cessation may be permanent.

Life is not directly threatened by the disorder, but when the shoulder girdle is affected so many muscles concerned in respiration or in expanding the chest are involved that imperfection in the former act and a falling-in of the chest walls prepare the way for pulmonary trouble, especially phthisis pulmonalis.   Most cases, however, die of some intercurrent disorder.

The diagnosis of progressive muscular dystrophy is not difficult in cases uncomplicated by some spinal disease.   The beginning of the affection in the muscles of the shoulder or pelvic girdle with the almost absolute immunity of the hand muscles from attack, distinguish the trouble from progressive muscular atrophy of spinal origin; hypertrophy, pseudo or real, is not seen in the spinal affection.

When the face is affected, as in the infantile form, the speech difficulty, from involvement of the orbicularis oris, may bring up the question of bulbar paralysis.   But in the myopathy the tongue, pharynx, larynx and masseters are not affected.

Treatment. Since it is held that progressive muscular dystrophy is but the manifestation of a defective development of the germ, to attempt its cure or arrest by medicines would be as futile as to endeavor by the same means to "cure" or change any other congenital defect. Gowers suggests from the variations in the time of appearance and its mode of extension that other influences may co-operate with the congenital tendency in causing the myopathy.

Muscular exercise seems to be a desideratum, for when it is stopped there is a quicker failure of strength (Gowers). In the pseudo-hypertrophic form the ability to stand is lost, through contraction of the calf muscles, before weakness causes the loss. Tenotomy is therefore advised, to let down the raised heels and enable the patient to stand, and the operation may be repeated with benefit if the necessity for it again arises (Gowers).

Of our remedies *Phosphorus* has been of undoubted service, and one case of cure (cessation of advance?) has been reported. In the writer's hands this drug in the sixth dilution (with daily applications of faradism over the affected areas) has been followed by undoubted improvement.

The general health of the patient should be looked after; its impairment will hasten the onset or accelerate the advance of the disease. Hence all the symptoms of the case should be taken into account when selecting a remedy, preference being given to the great antipsorics.

Progressive Neurotic Muscular Atrophy. This

rarely seen affection begins as a rule in the first or second decade of life, occasionally at a later period and it has been observed at birth (Goldscheider). It is a "family" disease, has appeared in several members of the same generation and has been noted in several successive generations. Males are attacked by it twice as often as females and the inheritance seems to be by the paternal line (Oppenheim).

It begins by wasting of the peroneal muscles, next the common extensors of the foot and the extensor of the great toe and later the calf muscles. In consequence gradual development of talipes varus or equino-varus appears and this is characteristic of the disorder. After some years the muscles of the hands are attacked, the thenar and hypothenar eminences wasting, as well as the interossei, giving rise in advanced cases to the "claw hand".

Both sides are affected, but not always in equal degree.

The muscles of thighs and upper arms escape for a long time.

Fibrillary tremor is frequently observed and the reaction of degeneration is present. Even in parts not yet attacked faradic contractility of muscles is slowed or even lost (Oppenheim).

The tendon reflexes disappear in the affected parts. Some hypæsthesia and, at times, pain, accompany the wasting.

The affection is believed to be a degenerative neuritis of the endings of the peripheral nerve fibres as a result

of defect in development. Whether the muscular involvement is secondary to the neuritis or is simply co-existent is not decided.

Prognosis for cure is *nil*, but as the disease has at times shown long periods of remission, treatment may be of service in retarding the advance of the affection. No case has been reported in our literature. Treatment must be constitutional after study of the whole case.

Neuroses.

Facial spasm. The muscles of the face are in such constant play in emotional expression and hence respond normally with such rapidity to cortical or subcortical impulses that it is not surprising that abnormal habits of activity are easily acquired. A not uncommon spasm is that of the orbicularis oculi (blepharospasm); it is often begun by voluntary contraction of the muscle in the endeavor to shut out painful impressions of light during photophobic conditions, or in the partial closure of the lids in myopes when looking closely, or by frequent fits of weeping, etc. It occurs in paroxysms aroused by some effort with the muscle, by a draught of air upon the eye, bright light, etc. The involuntary contractions are short (clonic), rarely prolonged (tonic), are not accompanied by pain and cannot be suppressed by the will. They are generally unilateral, but bilateral blepharospasm is not at all rare.

From a long existing blepharospasm the trouble frequently extends to the zygomatic muscles and then

to the muscles levator l. sup. et alæque nasi in some cases. The spasm may extend to the frontalis above, or as far as the platysma below.

Facial spasm is in most cases unilateral; the sharp quick contractions may occur in rapid series for a greater or less period, or may come singly at relatively long intervals. A paroxysm may be brought on by mental emotion, by exertion of muscles of face or jaws, by exposure to cold, etc.

The underlying cause is in most cases in the cerebral cortex; even where there is some evident peripheral cause whose removal gives relief, we must consider that cause to have been an exciting one only. The sufferers from this trouble are always neuropathic and not infrequently have some other nervous disorder, generally hysteria or neurasthenia.

The prognosis of the disorder is bad in neuropathic individuals; when a reflex cause can be found, its removal will give great help, but will not assure a cure if the trouble has existed for a long period of years. Blepharospasm dependent on ocular troubles is more amenable to local treatment.

Treatment of facial spasm by remedies is a thankless task if the trouble have existed for any considerable time. At the New York Ophthalmic Hospital the routine remedy for blepharospasm is *Agaricus musc.*, any ocular trouble being first relieved. At the writer's hands it has never done anything to relieve either blepharospasm or facial spasm. He has seen *Kalmia* cure for a time facial spasm, it being indicated by

the sense of stiffness about the eye of the affected
side. The trouble returned in a few months, but the
remedy failed then to help. Many of our drugs have
produced in the provings spasmodic twitching in the
face, but the selection should be made after study
of all the symptoms of the case. Before recourse is
had to surgery in extreme cases, it would be well to
try the effect of hypnotic suggestion.

Torticollis. Wry-Neck. An extremely obstinate
and annoying affection in which the head is turned
upon the neck in some abnormal position by tonic
spasm of certain neck-muscles. The muscles are the
sterno-mastoid, trapezius, splenius and those of the
sub-occipital region.

The sterno-mastoid is in almost every case affected;
the head is rotated to the opposite side, the chin
turned slightly upward, while the head is inclined
somewhat to the side of the overacting muscles. If
the upper part of the trapezius of the same side be
associated in the spasm, as it most often is, the in-
clination of the head to the same side is considerably
increased and the head is somewhat retracted, but the
rotation already existing is only slightly increased. If
the splenius of the opposite side be associated the
rotation is much greater. Both trapezii may be in-
volved and then there is retraction of the head; this
is a rare form and is termed retrocollic spasm. Other
combinations of muscles in the spasm occur with cor-
responding changes in the character of the abnormal
position of the head.

Distant associated spasms occur, such as spasm of the frontales when retrocollic spasm exists, or of the arm or face.

The disease most often appears in women and in early adult life. Its subjects are generally neurotic and often of neurotic heredity. It may come without any known exciting cause, but trauma, exposure to cold, over-use of the neck-muscles, exhausting occupation together with mental strain are common causes (a daughter nursed her mother during the latter's illness; the loss of sleep and the anxiety apparently caused an attack of wry-neck, which has persisted for several years).

There is no sensory disturbance, except some painful stiffness at the beginning. In some cases slight vertigo preceded the onset. The spasm begins in slight degree, often in one muscle only; it ceases and returns, increases slowly and extends, but it may be months before it reaches its height and becomes permanent. Cases have developed fully within a few days. In bad cases the spasm, with its periods of remission, becomes permanent, with no cessation except during sleep.

The muscles involved become hypertrophied after long continuance of the disease.

The pathology of the disease is not known, but it is surmised that for associated movements of the muscles involved, a centre, possibly in the pons, is the seat of some functional change. The writer considers the affection to be primarily cortical.

Diagnosis. Congenital wry-neck is seen in children. It is due to injury at the time of birth or to intra-uterine cause. In either case there is shortening of the sterno - cleido - mastoid muscle. The trouble does not show until the child is some months old, and later the shortened muscle is seen to be small and free from active contraction.

Disease of the tissues of the neck or spine may set up, reflexly, spasm. Examination will reveal such cause.

Prognosis for cure or even marked improvement is poor, yet undoubted cures have been effected. The writer recently received a letter of thanks from a patient who has now been free from the affection for more than a year. The remedies given were, however, prescribed for her general symptoms, and not merely for the spasm of the neck-muscles. *Lachnanthes tinctoria, Belladonna, Lycopodium, Lachesis, Nux vomica, Rhus toxicodendron* and *Sulphur* are credited with cures in homœopathic literature.

Habit Spasm. When in the company of a number of persons an observer occasionally finds his attention directed to some individual whose action is peculiar. This action may be some form of grimace (facial spasm), a shrug of the shoulders, a shaking of the head as if to settle the hat in place, or a movement as if to escape some annoyance from the collar, etc. The repetition of the movement at longer or shorter intervals shows the existence of a habit.

Habit spasm is most frequently seen in children,

but is by no means rare in adults. Its subjects are neurotic, often have some other nervous affection, and have a neurotic heredity.

The condition is probably of cortical origin, although its immediate cause may be reflex. Thus the winking spasm, or habit of winking much oftener than normal, may be set up by the presence of conjunctivitis or similar trouble.

Treatment of habit spasm must be that of the whole individual. In children hygienic and dietetic measures, together with physical education, will tend to strengthen the weak nervous system and distract the attention of the patient from self. No attempts to control the habit by parents or teachers should be made.

In the adult to improve the general health is about all that can be done by medicine, but the influence of suggestion may be effective; suggestion does not require hypnosis as a necessary condition— it may be carried by a placebo.

Epilepsy.

Epilepsy is a disease whose manifestations occur at varying intervals, and consist of loss of consciousness (in the ordinary sense of the word) with, usually, a general convulsion.

Convulsions similar in type to those of genuine or idiopathic epilepsy, with loss of consciousness, occur in the course of cerebral affections, with irritation of the cortical motor areas by gross lesions,

such as tumor, or as in dementia paralytica, pachy-
meningitis, syphilis, etc.; this condition is called
symptomatic epilepsy.

Reflex epilepsy is the state of recurring convul-
sions, with loss of consciousness, the exciting cause
being some peripheral focus of irritation. The cells
of the individual's cortex are probably unstable, but
not so much so as in cases of genuine epilepsy;
removal of the peripheral exciting cause cures the
manifestations usually. Among such causes are ir-
ritable cicatrices, affections of the digestive tract,
uncorrected errors of refraction, muscular anomalies
of the eyes, hypertrophies in the posterior nares, etc.

Jacksonian epilepsy is a symptomatic epilepsy,
and is due to an irritative lesion affecting part of
the motor area. The convulsive action begins in
the related peripheral part, as a thumb, a hand or
a foot, and in typical cases spreads to the rest of the
side and then to the other side; it may be, however,
limited to one part or to one side, and is not in-
frequently followed by temporary paralysis of the part
convulsed, and in such cases loss of consciousness is
not to be expected. Sensory disturbances, (paræsthe-
sias, etc.,) may accompany the spasm or may be the
only manifestation of the cortical explosive action.

Chronic alcoholism or the habitual drinking of ab-
sinthe sets up changes in the brain that lead to
epileptic manifestation, due probably to grosser alter-
ations in the tissues than those causing genuine
epilepsy. Trauma, blows or falls upon the head, may

cause a local meningeal or cortical lesion and eventually cause partial epilepsy, but in some cases the spasms cannot be distinguished from those of idiopathic epilepsy.

Lead poisoning, when long continued, is reported as having caused attacks not distinguishable from the genuine form.

The idiopathic disease is considered to be one of the degenerate states. In proof of this the following statements are offered. Heredity: the examination will show a history of some recurring severe type of nervous disease (epilepsy, migraine, etc.,) in a parent or near relative, or alcoholism, or insanity, or queerness, or "crankiness" in parent, or near relative; the patient frequently bears the evidences of degeneracy.

In the majority of cases the disease first appears in the second decade of life, the proportion being specially great about the period of puberty, a smaller though still considerable proportion occurring in early childhood in connection with the process of dentition, or following an attack of infectious disease, especially scarlatina. Cases developing after adult age are few and then are mostly of the symptomatic variety.

The frequency of the attacks varies greatly, yet it is almost a rule that at first they are separated by intervals of perhaps many months, but with recurrence the interval lessens. In a case well established in point of time, the average interval is in my experience three or four weeks. Often the attacks are grouped, several coming on within a week or two, being fol-

lowed by a period of freedom for some months. Daily attacks are not rare, especially when of the lighter type.

The attacks occur more frequently during the waking hours than during the night-sleep; in some instances they happen only during the latter and are then classed improperly as nocturnal epilepsy. In women the spasms tend to appear at or just before the menstrual period.

The occurrence of an attack can not infrequently be traced to indigestion, excesses of different kinds, strong mental emotion, etc.

Premonitions of the attack are felt and in course of time recognized as such by the sufferer in many instances; they are conditions of malaise of many kinds, irritability, sense of fatigue, mental depression, etc., and may exist from a day or two to a week or two, before the outbreak.

Immediately preceding the attack and properly a part of it, certain warnings of its oncoming are felt in a large proportion of the cases.

This warning is generally some paræsthesia (often the sensation of a breeze blowing on some part— hence the term *aura* which has been applied to all the forms of warning), or an indescribable sensation beginning in the region of the stomach and ascending to the head, on reaching which the attack is fully on.

The attack begins as a sudden onset of unconsciousness, the patient falling from the upright position or from the sitting posture to the ground, and immedi-

ately a tonic spasm affects the whole body so that the individual is stiffened, the back being more or less arched; the head is retracted or turned to one side, the eyes often deviated to the same side, the upper limbs more or less flexed, the lower ones extended. In the beginning of the tonic stage the patient utters a cry as a result of the sudden spasm of respiratory and laryngeal muscles; respiratory action ceases, and the face, which at first is pale, becomes flushed and then cyanosed. This tonic stage lasts from a few seconds to half a minute or so, and is then succeeded by the stage of clonic convulsion in which the limbs are affected by alternating flexor and extensor contractions of considerable amplitude.

The transition from the tonic to the clonic convulsion, while rapid, is not abrupt. Intermediately a quivering or trembling in the muscles, or twitchings especially in the face are seen and then the clonic stage begins.

The movements have not the character of purpose, but the patient may be injured by the violence of the muscular spasms; the tongue is often bitten during the masseters' contractions and the accumulated saliva which usually appears as froth at the lips, is tinged red by the blood from the thus wounded tongue. The face is distorted and the eyeballs may be actively in motion; the pupils are dilated and do not react to light. The body temperature is slightly raised and the pulse somewhat accelerated. During this stage the bladder is frequently evacuated and occasionally

fæces or semen discharged. The convulsion may cease only to be renewed, or may gradually lessen until it finally disappears.

The clonic stage lasts a few minutes only, indeed the time limit of the whole attack up to the cessation of spasm, is of importance in diagnosis.

The next stage is that of sleep or at times apparent coma with stertor. In it the patient usually does not respond to external impressions. Its duration is variable; when the patient awakens, it is usually with a headache (that in many instances only disappears during the next normal sleep) and a general aching and tired feeling.

The preceding description applies to the severe attacks (*grand mal*), but many cases have only mild attacks (*petit mal*) in which indeed there may be no motor phenomena. The lightest form of petit mal is a sudden stoppage of mental activity, as if in a "brown study", lasting but a few moments; at its cessation the patient resumes his previous occupation, takes up the thread of conversation, often indeed not knowing that anything has happened. The pale face, the fixed stare, the dilated pupils and the unresponsiveness to remarks, tell to the acute observer the true state of the case, while in the existence of twitchings of the muscles of face, lips or tongue, of the eyelids or of the extremities, even an uninterested observer will recognize the trouble. The attack of petit mal is often preceded by an aura.

In some cases the individual simply falls uncon-

scious and remains so for a few seconds without any spasm, or falls asleep to all appearance and wakens after a short time evidently surprised. This form is known as narcolepsy.

Procursive epilepsy is that form (rare) in which the patient is apparently seized by an impulse to run or walk forwards for some distance (rarely backward), or does some other action—all as if in a dream and in unconsciousness; he may go from this state into an ordinary epileptic convulsion or may return to ordinary consciousness.

The loss of consciousness may be an alteration of the patient's consciousness of his real self, and of his relations to the outside world, but in which state he may act as an ordinary individual and may commit crime. This state of altered personality is not accompanied by convulsions, and when the patient emerges from it, he is without memory of his acts while in it, or there may be only a dim and fragmentary remembrance. This condition is known as the psychical equivalent of epilepsy.

The state known as psychical equivalent of epilepsy, already mentioned, may come on at the close of an ordinary convulsive attack, in which case it is termed post-epileptic insanity, or it may be the only manifestation of the disease. In either instance the patient may be apparently delirious or maniacal, or may be as if in a dream. The forensic importance of this form will be understood when it is seen that crime has been committed while in this state, and, that as

the consciousness of the individual is suspended during it, his legal responsibility is also absent; the difficulty of proving the absence of ordinary consciousness is very great when the ordinary attacks of epilepsy have not already occurred.

What is known as the status epilepticus is the occurrence of a series of convulsive attacks in rapid succession, indeed passing directly from one convulsion into another. The condition is a dangerous one, is attended by increase of temperature and may end fatally.

Between the attacks, especially if the interval is long, the patient is sound in body and mind; in cases in which the attacks are many and at short intervals, there follows some mental deterioration, and in the young a weakening of the moral sense. That epilepsy is one curse of genius is no longer held, and the fits with which Napoleon, Mahomet and others of great fame were afflicted, are considered by Möbius to have been probably hysterical.

The epileptic, at least such as suffer from the severe attacks, live in an atmosphere of danger. Falls into a fire or from a height, or before an oncoming vehicle or railway train, have repeatedly occurred. Apart from such dangers, the disease process itself undermines the resisting powers of the organism.

The pathology of epilepsy is still a terra incognita. Changes in the brains of those who had epilepsy during life have been too varying to justify acceptance of any one as the underlying cause, while the theory that certain layers of cells in the cortex are

predominantly diseased, needs the support of more
extended observation. It seems certain, however, that
the cerebral cortex must be the seat of the disease.
The diagnosis of genuine epilepsy is to be made by
a knowledge of the symptoms mentioned, by the
exclusion of any cause of the symptomatic form or
of reflex spasms (eclampsia infantum). In a hys-
terical convulsion the pupils respond to light, the
spasm is largely made up of purposive motions, such
as fighting, struggling, thrashing the body up and
down on the bed, the patient often talks or screams,
and convergent strabismus may be present, while the
tongue is not bitten although the lower lip may be.
The hysterical convulsion usually lasts much longer
than an epileptic one. Sometimes a minor epileptic
seizure passes into a hysteroid convulsion like that
just mentioned, and the epileptic stage may be diffi-
cult to ascertain unless the physician is present and
examines the pupils.

A minor attack may be mistaken for a fainting fit
if no spasm or twitching is present.

Nocturnal epilepsy (occurring during sleep) is almost
always genuine epilepsy ; if the individual sleeps
alone, the existence of the trouble may not be dis-
covered for a long time.

Auditory vertigo may be mistaken for epileptic ver-
tigo, especially in severe cases, when the patient falls.
The evidence of ear symptoms, especially tinnitus and
deafness, will clear up the doubt ; Gowers has, how-
ever, seen the two conditions associated.

The prognosis. As regards the epileptic seizure, apart from the danger when it occurs in some position of peril, or when during it the patient's face is turned into a pillow or the bed-clothing, it rarely ends fatally, except in the status epilepticus. As regards recovery from the disease, the prospect is very poor. Gowers does not use the word cure or recovery in this connection (except in some cases in early life where after appearing, the fits disappear at the age of four or five). He considers that the only way known of curing the disease is by keeping the fits away long enough to permit the morbid tendency to subside. That the fits can be held in check in many cases by continued doses of bromide cannot be denied, but Gowers considers that the drug should be exhibited in the same dosage for two years after the last fit, and during the next year the dosage is to be gradually diminished to nothing.

Möbius holds that treatment, although of great importance, cannot cure the disease in the real sense of the word. Of bromide he says that it is indispensable and that so sure is its action in lessening the excitability of the cortex, that its use is of even diagnostic value. When by its use the attacks are not lessened they are probably hysterical. Gowers, on the other hand, says that it is far more common for bromide to have no influence in the case of petit mal than in the severe attacks.

It will thus be seen that the prognostic views and percentages are largely affected by bromide as a factor,

and the statistics given by Gowers and others should be headed by a line reading "Under treatment by bromide."

Nevertheless, the disease is curable only with difficulty, and homœopathic treatment applied according to the method of strict individualization will show a far greater percentage of cures than will the bromide or opium treatment, or combination of both, or any other drug or aggregation of drugs. Homœopathic statistics have not been as yet collected and analyzed, and undoubtedly many cases of cure of epilepsy have been reported by physicians of all shades of therapeutic belief, which would not bear strict diagnostic tests. Möbius does not believe in the possibility of a real cure, but he is optimistic in claiming possession of a diagnostic drug; Gowers speaks of cessation of the attacks as the highest aim possible, but if we accept Möbius' diagnostic procedure a good many of Gowers' cases of petit mal must have been hysterical.

Gowers' probabilities in prognosis are as follows: The prospect of arrest is slightly better in males than in females, better if the disease begins after twenty than before, better the shorter the duration of the disease (being greatest when it has existed less than a year), better the longer the interval existing between the fits (very poor if fits occur daily), better if the fits occur only during the sleeping or the waking state than if they occur in both, better if there is no considerable mental change, better if the attacks are all of the severe variety than if there are minor seizures,

and better if the attacks are preceded by an aura than if they occur without warning.

Epilepsy due to organic disease, as well as the insanity following epilepsy, is hopeless of cure.

The percentage of epileptics that become finally insane is given variously in different statistics; it is as low as 3 per cent. in some French statistics, and as high as 10 per cent. in some American ones.

The treatment of epilepsy as a disease is largely a matter of hygiene. Mental and physical comfort (not enforced idleness), with some pleasant, not dangerous, occupation, the avoidance of mental or physical over-exertion, food of easy digestion, with moderation in the amount of meat, are to be prescribed. Alcohol is to be forbidden; coffee and tea had better be proscribed. Indigestion, as shown by foul breath and coated tongue, is often the forerunner of a seizure, generally with constipation as an accompaniment.

The epileptic subject is always in personal danger and should not go out into a city's busy streets without an attendant, while the peril of unprotected platforms or bridges, nearness to open fireplaces, the ordinary full bath in the regulation tub, need only to be mentioned here. Swimming (and bicycling) should be absolutely prohibited.

That the homœopathic treatment of epilepsy has resulted in cure is, in the writer's opinion, an undoubted fact, but the number of such cures would have been largely increased could the treatment have

been continued in all cases. Cases come to the physician, take the remedies for a few weeks or a few months, and then, not being wholly relieved of the attacks, cease attendance. The writer prefers not to take a case of epilepsy unless with the understanding that he is to have full charge of it during a period of two years. Another cause of ill-success is the selection of remedies upon the indications observed during the fits, such as the color of the face, etc., instead of ignoring such points until a study of the whole patient has been made.

Jahr found that most good was done in this disease by the use of *Sulphur, Calcarea, Lycopodium, Causticum, Cuprum, Silicea* and *Lachesis*. *Belladonna* and *Sepia* have been credited with good results. *Absinthium* has been much praised for its influence when the fits are frequent in the 24 hours. Reports of cures, or at least of cessation of the fits, under the action of *Œnanthe crocata* tincture and also *Solanum Carolinense* tincture, have repeatedly appeared in the journals. In the writer's experience the latter has done nothing, the former helped in one case, reducing the number and severity of the attacks, but causing headache, which finally compelled the withdrawal of the remedy. In one case, *Borax* 3x, four times a day, caused marked improvement, but the patient disappeared after three months' treatment. Positive results seemed to follow the use of *Bufo* 3, not in one case only, but in several. *Lachesis* 30 helped a case sent to the writer for opinion, and kept the fits away for

several months; a recurrence taking place, a renewal of the prescription was effective, but the outcome of the case is not known. *Melilotus alba* has been recommended by Dr. Bowen as a specific for all kinds of fits; in the writer's hands it has not helped in epilepsy. Many other remedies have been praised by different observers in the treatment of epilepsy, but enough has been said to show that thorough study and sharp individualization are necessary, in order to make any lasting and beneficial impression upon the disorder.

In cases that have been treated carefully by homœopathic measures for two years without result, it would seem futile to attempt longer a cure; and when no noteworthy lessening in the number of the attacks occurs, it would also seem to be justifiable to permit the use of bromides if they will keep the fits at bay without doing serious injury — in other words, palliative and antipathic treatment for relief, not homœopathic for cure, is then in order.

Migraine.

Hemicrania. This affection is a constitutional vice whose manifestation is the occurrence at longer or shorter intervals of a peculiar headache accompanied in most cases by gastric disturbances and often by sensory irritative phenomena.

The heredity is proven, first, by the fact that in many cases the parent had the same trouble or some other severe neurosis and, secondly, by the fact that

parents afflicted with migraine at times transmit to their offspring a still worse disease, *i. e.*, epilepsy.

Migraine generally first appears about the period of puberty, but may begin in early childhood; rarely after the age of 30.

In general the attack is preceded by some condition of malaise, drowsiness, pressure on the head, irritability, etc. It begins, as a rule, in the morning, as headache located on one side and mostly about the eye, but it may extend across the whole frontal region or back to the occiput, or may involve the whole head. In some cases it begins in the occipital region.

The pain itself may be dull and not interfere with the patient's occupation, but oftener it increases to an intense pressing or boring that is made almost intolerable by motion or by any sensory irritation. Hence, the patient keeps in bed in a darkened quiet room.

With the pain there is great weakness and nausea, often intense, and leading to vomiting. The vomiting may be so often repeated that bile is eventually brought up and thus the name "bilious headache" has come to be applied to the attack.

The face is in the majority of cases pale and cool; the pulse is small; in some cases the face is red on the affected side. There has been an endeavor to make two classes of migraine, one with pale face (due to spasm in the arteries), the other with redness (due to paralysis), but it is now believed that the condition of the circulation in either case is a secondary one.

After reaching its height the pain gradually diminishes and the sufferer is able to sleep, awakening, as a rule, well. In some instances the attacks last but a few hours; in others two or even three days.

In the cases having associated sensory disturbances the most frequent are the visual ones. There is often a so-called blindness; it is rather a darkening before the eyes, likened by the sufferer to a fog or mist. Or he sees a bright curve or irregular line or scintillations, usually in one part of the visual field. The color of these appearances may vary. In one case the appearance was that of a sheet of some dimly seen material hanging at the left of the patient and having a broad yellow metallic band at the bottom. In another there was the appearance of a snowstorm before the eye, the flakes glittering. Often the figure is that of a zig-zag in some bright metallic coloring, to which the term fortification-spectrum has been applied. These visual hallucinations last but a few minutes, as a rule, and are usually the immediate precursor of the pain. The clouding of vision may be hemianopic. Temporary aphasia has been observed also as a preliminary symptom.

Paræsthesia of one arm or one side, or even of both sides (face and tongue) have been noted and also hemiparesis.

Migraine has been reported in which during the early part of the attack paralysis of one or more of the eyeball muscles occurred.

Attacks of severe vertigo and episodes of mania or

of confusional insanity, may appear instead of the regular headache.

The intervals between attacks vary somewhat and yet the latter have a certain regularity; they are especially apt to occur at the time of the menses. They may be brought on by the use of certain articles of food owing to idiosyncracy of the patient. Thus the use of sugar has more than once been a cause, while whipped cream in any guise has been an exciting cause in one case. Possibly the time at which such articles act thus may have been coincident with that of an oncoming attack. Also a fit of anger or other strong emotion, or travel in a close railway car has precipitated an attack.

Reflex causes such as errors of refraction, heterophoria of eye-muscles, hypertrophies and other abnormalities in the rhino-pharynx, when present, may be actively exciting causes of attacks of hemicrania.

The course of the disease is not much influenced by anything; it continues during life in many instances, but in a large fraction of the cases it becomes less assertive in the number and violence of the attacks between the ages of 45 and 50 or may even disappear. Change of climate has proven so beneficial in one case that a cure was supposed to have taken place; but return to this country was followed by recurrence of the attacks.

The characteristics of the first attack usually reappear in the subsequent ones, but sometimes two sets of symptoms alternate in two attacks.

The pathology of migraine is a matter of surmise. The chief views concerning it are the vaso-motor theory, angio-paralysis and angio-spasm in or about certain nerve centres; the theory of "nerve storm" which may mean anything; that of irritation of the fibres of the fifth nerve within the skull with secondary affection of the vagus nuclei. None deals with the phenomenon of periodical recurrence.

The treatment of migraine involves two problems; first, the relief of the attack and second, that of the underlying general condition.

In the former the remedies should be selected according to the special indications then present. The side affected (right or left), the quality of the pain, the influence of heat or cold, the position of the patient (lying upon the affected side or *vice versa*), in fact all the conditions of amelioration and aggravation, should be taken into account. The special symptoms introducing the attack, such as dimness or blurring of vision, colored spectra before the eyes, take high rank among the indications for remedies.

Chief among the remedies proving beneficial in the treatment of the attack are *Belladonna, Ignatia, Argentum nitricum, Iris versicolor, Stannum, Sanguinaria, Spigelia anth.* and *Sepia.* In any case of this distressing headache the selection of a remedy should not be made hurriedly.

Ferrocyanide of potassium 30, in water, repeated every hour mitigated the severity of the pain in one case. *Cyclamen* 200, given in the same way during

several attacks seems to have cured the patient, no return of the trouble having occurred during four years. *Argentum nitricum* 30, given during an attack was followed by the cessation of the trouble for two years.

During the interval the patient should be examined for any possible exciting cause for the attacks and such cause should be removed either by internal remedies or by such measures as the correction of ocular defects, etc.

The coal-toar products such as acetanilide and phenacetine have been resorted to as palliatives. They may be of great value in some cases, but the writer has seen them act promptly the first time and fail utterly in subsequent attacks. In one case the attack while partly relieved was much lengthened, and in another the attacks seemed to increase in frequency after their use.

Chorea.

The chorea of Sydenham, or chorea minor, is a rather common affection, seen mostly in those between the ages of five and fifteen, and in girls more than twice as often as in boys, while after the latter age the cases up to 25 years are almost always females.

The cause of the disease is unknown, yet its association with rheumatism and endocarditis has been so often observed that a common influence for both has been accepted. The proportion of cases in which articular rheumatism has an apparently causal

relation to the outbreak of chorea, has been estimated by American writers as from 15 to 25 per cent., but this proportion is increased when we consider that endocarditis may exist without any other evidence of rheumatism. It occurs relatively frequently during pregnancy, usually in young women and during the first pregnancy; it occasionally follows one of the infectious diseases.

Fright is the exciting cause in about 25 per cent. of the cases, and where injury is assigned as a cause the existence of fright cannot be excluded. In the majority no special exciting cause can be found; in some instances the trouble seems to depend upon intestinal or other reflex cause.

The onset of the disorder is gradual in most cases. The child is noticed to be restless, or to "drop things"; at school the writing becomes irregular and for this and the restlessness it is often unjustly punished. Later a hand or arm is thrown into sudden action, pronated or supinated, extended or contracted, a finger or thumb is actively moved, the mouth or forehead, or other part of the face twitches, a shoulder is jerked up, a foot or the whole leg is moved in some unexpected direction, etc. The movements are sudden and rapid, without rhythm, and often occur during some purposed action with which, indeed, they interfere. In bad cases walking may become difficult or impossible from such interference, or the patient cannot help herself at table, etc. Speech may be rendered difficult owing to

implication of the muscles of tongue and lips; similarly, swallowing or phonation may be interfered with, while, when the respiratory muscles are involved, respiration may be spasmodic and irregular.

The strength of the irregular muscular contractions varies in different cases; in some they may be slight and confined almost to the small muscles of the hand; in others the whole body may be flung about. The movements cease during sleep, but sleep is delayed or at times even prevented by the motor unrest.

In children there is often mental and emotional irritability, and they not rarely waken from sleep in fright. In young women at the height of the disease, delirium with hallucinations and maniacal excitement may appear.

The tendon reflexes are not altered and no sensory disturbance occurs, except as part of a rheumatism, that may exist. Weakness of the limbs is often present. Many of the cases are anæmic, and in bad cases the interference with feeding and sleeping and the exhaustion from the almost constant activity of muscles lower the strength.

Paralysis is not present, but in some cases the disease begins as a difficulty in using one arm, which difficulty becomes, later, total inability. An occasional muscular contraction and its increase with a corresponding decrease of the pseudo-paralysis, show the true nature of the affection.

The average duration of chorea is between two

and three months, but not infrequently an attack will continue for six months or so; the writer has seen a case that had persisted for more than two years.

In a considerable proportion of the cases a relapse occurs during the next year; in a few instances a second relapse is seen, but rarely more. The relapses occur only in children and in chorea gravidarum; on the other hand, chorea in the adult may become permanent.

The prognosis is good as regards life, death being rarely the result, and then generally in adults; those dying, have, in most cases, the cardiac affection so often present.

The diagnosis of chorea is not difficult. The character of the movements already stated usually suffices for the purpose; some difficulty may be met in separating habit-spasm in its beginning from an oncoming chorea, and choreiform movements are seen to follow hemiplegia in the young—the history, and rigidity of the affected side, will then show the true nature of the trouble.

The pathological anatomy of chorea is unknown, the mortality of the disease being very small, and the cases coming to the autopsy table being generally severe and complicated. The findings have not been constant. The generally accepted view is that the disease is an affection of the brain in which the motor cortical areas and perhaps the large basal ganglia are specially involved.

The treatment of chorea is a matter of studying

out the remedy, and often a succession of remedies
will be required. The writer has had successes with
Ignatia, Tarentula, Cimicifuga and *Pulsatilla.* In one
case, after treatment of some weeks without result,
a re-study of the whole individual led to the selection
of *Calcarea carb.*, which, in the 200th potency, cured
promptly. He has never obtained any result from
Mygale. In allopathic hands he has seen remarkable
results from the use of Fowler's solution, but *Arseni-
cum*, in homœopathic preparation, as low as 2x, has
failed him; it may be that much of the effect of
Fowler's solution is due to the potash it contains, and
hence that *Causticum* ought to be used by homœopaths
more frequently in this disease. Goodno and Bartlett
use *Agaricine* 2x as a routine remedy, and claim bril-
liant results from its use. Other remedies recom-
mended by different observers are *Veratrum viride,
Cuprum aceticum, Belladonna, Zincum* and *Sulphur. Gel-
semium* tincture has cured several cases. In any case,
the selection of the remedy must be made by study
of all the symptoms.

The term **electric chorea** has been applied to three
different affections. Henoch has described a kind of
chorea appearing in children; its chief characteristic
is the lightning-like rapidity of the muscular con-
tractions, otherwise like those of ordinary chorea, and
affecting especially the muscles of the nape and
shoulder regions.

Bergeron uses the term electric chorea to designate

an affection seen in young persons from 7 to 14 years
of age, in whom the contractions are lightning-like in
rapidity, involving groups of muscles and affecting one
or more members.   Thus, the head may be thrown
in some one direction, the arm abducted or the fore-
arm flexed, etc., with a certain rhythm.   The con-
tractions cease during sleep.   Effort of the will to
restrain them seems rather to increase them.   The
condition has a good prognosis, and Oppenheim con-
siders it to be difficult to distinguish the affection from
hysterical chorea.

A peculiar disease, found in upper Italy, has been
described by Dubini, under the title electric chorea.
It may begin at any age with pain in head and back,
to which are soon added contractions, lightning-like
in rapidity, in one arm, one side of the face or,
indeed, of the body, or affecting the whole body.
Epileptiform attacks occur and may be limited to one
side; paralysis and atrophy are seen and the temper-
ature may be considerably increased.   After some
weeks or months death occurs in most cases, usually
by cardiac paralysis or by coma.   Some infectious
material is considered as the cause (Oppenheim).

Hereditary chorea, or **Huntington's chorea**, is an
affection first observed in Long Island.   Cases of it
now exist in Connecticut.   It is inherited, affects both
sexes and does not appear until about the 40th year.
Its first manifestations are twitchings in the face;
later, the arms and legs are affected.   Progressive
mental change occurs until dementia supervenes.   Post-

mortem, pachymeningitis, leptomeningitis, encephalitic processes, especially in the cortex of the motor areas and in the white matter beneath, have been found. The condition is incurable.

Paralysis Agitans.

Parkinson's disease. — The shaking palsy. This disease appears generally after the fiftieth year, rarely after seventy or before forty.

Its characteristics are a special tremor, a peculiar rigidity of muscles, a typical position of the body and of the thumb and adjoining fingers, and definite alteration of the gait in walking.

The onset of the disease is slow, the first symptom being some slight tremor in one hand, with or without a sense of weakness or stiffness. These first symptoms do not continue, but reappear under the influence of strong emotion or fatigue. In time they become permanent and later extend to the lower limb of the same side. Thence they extend to the other arm and leg. The tremor is rather coarse, having four or five oscillations per second, and is peculiar in that it is present during rest and not during voluntary effort; it may increase somewhat at the beginning of a voluntary motion, and in a late stage of the disease may continue during the latter. It is increased after muscular effort and during emotion and when the patient is conscious of being observed. The tremor ceases during sleep.

The special rigidity of muscles is a myotonia with

more or less contracture dominating the flexor muscles
and, in consequence, giving rise to a typical position
of the body (when standing) and of the hand.

The head is inclined forward, the body inclined for-
ward, the lower limbs are bent at the knees; the
fingers and thumb may take the penholding position
or may be simply in the position of rest, but in either
case the tremor affects the fingers and thumb inde-
pendently, causing the thumb and forefinger to move
as if rolling pills.

The position of the body in standing, as described,
is such that a line from the centre of gravity falls
in front of the feet, and consequently, in walking
the patient would fall, did he not hasten his pace,
which finally becomes in typical cases a run, and
when he can go no faster he brings up against a
door post, a wall, tree or some other object, from
which he can make a new start. This hastening in
his rate of progression is termed *festination.*

The myotonic condition of muscles appears on
attempting a movement. On starting to walk he may
have to make two or three efforts to overcome it,
and then he almost falls (propulsion); in some cases
he is forced to take two or three steps backward
before he can go forward (retropulsion). Rising from
a sitting posture is accomplished only after repeated
effort and by great exertion; to turn over in bed may
be extremely difficult or impossible without assistance.
Of this peculiarity the patient says that his feet are

heavy or are anchored to the ground, or that he feels as if fastened in the chair, etc.

As the disease advances the speech becomes affected and the words are hurried; the voice becomes thin, high, pitched and wavering. These symptoms are not always present.

The nutrition of the patient is often good, the face plump and of blooming aspect. In such cases the fullness of face obliterates its lines and it is without expression.

The tendon reflexes are normal or occasionally exaggerated. Sensibility is not altered, but pains, rheumatic in character, may be present; an overpowering sensation of heat torments the patient in some cases, and there may be excessive secretion of sweat.

The mental state is not altered unless in advanced stages, when the persistence of the annoying symptoms causes emotional irritability, with some tendency to depression.

The disease is slowly progressive, two years or more elapsing before the full manifestations are evident. Occasionally there is rapid progress, especially when the apparent cause is severe mental or physical shock. After the affection is fully established, in many cases no increase in the intensity of symptoms may be observable for a long time, but with advancing years the increase in the muscular rigidity and continuous tremor render the patient more and more helpless, until he finally

becomes bedridden. When the muscular rigidity becomes very great the tremor disappears. In some cases the rigidity appears without tremor; such cases are termed paralysis agitans *sine agitatione*, and are recognized by the characteristic posture, etc.

The prognosis as regards life is good; death occurs from exhaustion or from some intercurrent disease.

The pathology of the disease is as yet unknown; effort has been made to see in the different results obtained from post-mortem microscopical investigation, evidences of early senile degeneration.

With a hemiplegia, tremor may develop in the paralyzed limbs, simulating the tremor of paralysis agitans. The history, the increased tendon reflexes, the evidences of paralysis, will correct any doubts. Senile tremor affects chiefly the head, begins much later in life and is without the typical position of body or hands noted in paralysis agitans.

The treatment of paralysis agitans is hopeless as regards cure; homœopathic remedies have not, in the writer's hands, been of any service in the affection. Antipathic treatment by hyoscyamia in so-called physiological doses may overpower the spasm and tremor and give the patient some relief.

Tetany or Tetanilla.

Tetany or tetanilla is a disease characterized by the occurrence usually intermittently, of bilateral tonic spasms affecting certain groups of muscles mostly in

the upper extremities, accompanied by pain and generally without loss of consciousness.

The disease has appeared epidemically in certain parts of Europe, as in Paris and Vienna, while it is almost unknown in Northwestern Germany. In this country it is extremely rare.

It seems to affect the working class only and in v. Frankl-Hochwart's tables one-half of the cases (males) were shoemakers and one-quarter tailors. In 88 per cent. of these the disease appeared between the ages of 14 and 25; in less than 1 per cent. it appeared after 50.

It occurs after infectious diseases, after complete extirpation of the thyroid gland, during the nursing period in the mother or child, in the latter in connection with intestinal disease or with rickets, and in association with dilatation of the stomach.

The existence of small epidemics has led to the view that it is an infectious disease *sui generis*, but no special germ has as yet been found. Its relation to infectious fevers, to toxines or ptomaines as in the foregoing instances, are in line with its recorded occurrence after poisoning by alcohol, ergotin, chloroform, lead, phosphorus, etc. We can only say that a specific origin is as yet not proven. Fright and exposure to cold have been followed by tetany.

The onset of the spasm is preceded, usually, by paræsthesias or even pain in the fingers and hands and later a sense of stiffness. Then the interossei and the adductor muscles of the thumbs contract, with

the result that the fingers are flexed at the metacarpo-
phalangeal joints and extended at the others while
the thumb is pressed against the last phalanges of the
first and second or second and third fingers, the hand
thus assuming the position of the accoucheur's hand
preparatory to its entering the uterus for the opera-
tion of turning, or it may have the position used in
holding a pen. In some case the fingers are flexed
at all the joints, the thumb being thrust between the
first and second fingers. The hand is flexed upon the
forearm and often the forearm upon the arm. In the
lower limb the spasm is a plantar flexion of the sole
and toes, but the foot may be extended upon the leg
and the leg upon the thigh.

The trunk muscles are rarely affected; the masseters
and the muscles of the neck may be at times con-
tracted. The eye muscles may be involved causing
temporary strabismus or spasmodic closure.

The myotonic condition seen in Thomsen's disease
has occasionally been observed in tetany, as it has
also in hysteria, in multiple sclerosis and in muscular
atrophy — (v. Frankl-Hochwart).

Certain tests of diagnostic value have been dis-
covered. They all depend upon the existence of
hyperexitability of motor and sensory nerves. When
pressure is made upon the main nerve trunk of the
affected part continued spasm of the part can be
brought about. (Trousseau's symptom.) If a motor
nerve (best in the face) be mechanically irritated as
by a tap or by stroking, contraction of facial muscles

follows. (Chvostek's symptom.) If a motor nerve be tested with the galvanic current it will be found that cathodal closure contraction occurs with weak currents while cathodal closure tetanus is easily produced and anodal opening tetanus in almost all cases. (Erb's test.)

Hyper-irritability of the sensory nerves to both mechanical and electrical stimulation is observed in many cases. Light pressure upon the supra-orbital nerve, for instance, causes an out-streaming paræsthesia in the distribution of the nerve. With the galvanic current the spreading sensation that is only produced normally by strong currents, appears here with light ones. The hyper-sensitiveness is known as Hoffmann's symptom, but is not confined to tetany.

The affection may last for weeks or months, or may be over in a few days. The individual spasm may continue but a few minutes, or it may be a few hours or days.

Tetany has been seen in association with myxœdema.

The diagnosis is usually easy, but the possibility of hysteria having the same type of spasm and even of showing Trousseau's symptom is seen in a case reported by L. Minor (Neurologisches Centralblatt, 11, 1896). A case reported by the writer in N. A. Jour. of Hom'y, 1888, was believed to be genuine tetany until the application of faradism, made because of alarming symptoms, brought about complete relief of all the symptoms. The subsequent course of the case is unknown

to the writer. In Minor's case there had been all the
signs of genuine tetany, but some disappeared and the
hysterical manifestation mentioned seemed to be due to
suggestion.

From tetanus the disease is differentiated by the
intermittency of the spasm, by its beginning in the
muscles of the hands, and by the late occurrence of
trismus, when this occurs at all.

Tetany without spasm has been observed; that is
to say, paræsthesias and pain, especially in the hands,
are experienced, and Chvostek's and Erb's symptoms
are present. The state is termed tetanoid and may
pass over into the spasmodic form.

The prognosis for cure is good in most cases, except
those following thyroidectomy or dilatation of the
stomach. Cases following an infectious fever recover
rapidly.

Myotonia Congenita.

Thomsen's disease. A rarely occurring disease and
hereditary. In the family of Dr. Thomsen twenty
cases have appeared in four generations (Oppenheim).
The affection may begin in early childhood, at puberty
or even later.

The peculiarity of the disorder consists in the in-
voluntary continuance of a voluntarily begun muscular
contraction. Thus, the patient grasps the hand of
another and then is unable to let go; if he closes his
eyes he is unable for a time to open them; with the
larger muscles the tonic spasm seems to set in with

the beginning of an effort, so that rising from a chair or starting to walk brings on a feeling of heaviness and restraint that lasts for some seconds. Repetition of the effort, however, seems to limber up the muscles after some trials, and then they act in the ordinary way.

The tonic state is heightened by mental excitement and by cold. In some cases the trouble is confined chiefly to the limbs, in others it may affect all the voluntary muscles. The muscles are unusually voluminous, but their strength is less than their appearance would indicate. The electrical reaction of the nerves is not altered, but that of the muscles is increased. To faradism the muscles show tonic contraction, with continuance of the contraction after the current has ceased; to galvanism they show anodal closure contractions, usually with continuance after the current ceases. Repetition of the procedure at short intervals will bring about a normal reaction. With strong stabile currents Erb has caused rhythmical waves of contraction in muscles, passing from cathode to anode. In some cases this phenomenon could not be obtained. In what direction the congenital fault lies, whether primarily in the muscular or in the nervous system, is as yet unknown. There is no abnormality in sensation, the reflexes, etc., but complications, such as epilepsy, psychical changes, etc., have been observed. The condition is life-long in duration, but remissions are said to have occurred.

Related to, yet distinct from, Thomsen's disease, is

paramyotonia congenita, described by Eulenburg. It was traced through six generations of one family, and in some of the cases the symptoms showed immediately after birth. The chief symptom is the occurrence of the tonic spasm, not from voluntary contraction of muscle, but from exposure to cold, at times even in slight degree. The spasm lasts from a quarter of an hour to several hours, and after its cessation there follows some weakness for a time. The orbicular muscles of eyes and mouth are mostly affected, and the arms more than the legs. The electro-excitability of the muscles is lowered, but tetanic contractions are more easily produced by the current than normally.

Gowers describes ataxic paramyotonia as a union of persistent spasm similar to the temporary one of Thomsen's disease, with ataxia, weakness and anæsthesia, especially in the hands. Electrical irritability of the muscles was unaltered. The trouble began at the age of 40, but the outcome of the case is not known.

Jolly has reported recently a case similar, in all respects save one, to Thomsen's disease. The patient, a hard-working man, suffered repeated chillings, and at the age of 42 found that first his right and next his left hand became weak and would not obey him, owing to the tonic spasm that developed on closing them. The arms and legs became affected. The electrical reactions were as in Thomsen's disease, save that Erb's waves could not be produced. The

variation from the former disease was the presence
of distinct atrophy of the muscles of the right thenar
eminence.

Myotonia, as a symptom, has been repeatedly seen
in paralysis agitans, and an exquisite case of this has
been reported by the writer (Hahnemannian Monthly,
1895). The patient, on attempting to rise, could not; he
felt as if he weighed a ton; on attempting to walk, his
feet felt as if anchored to the floor; to turn over in
bed at night was impossible, etc.

Other and lesser myotonic symptoms have been
seen by the writer in two hysterical cases. In one,
the patient took hours at a meal, because she could
not lay down her knife or change her spoon or cup
until after many efforts.

Jolly remarks upon the extraordinary resemblance
between the muscular symptoms observed in poison-
ing by veratrine and those of myotonia.

Hysteria

is a condition rather than a disease; it is the ex-
pression in different ways of a faultily-planned nervous
system, and hence it is a form of degeneracy. Janet,
Blocq, Möbius and others term it a psychosis, the
latter author describing it as an insanity characterized
by a weakening of the faculty of psychological syn-
thesis, combined with a narrowing of the field of
consciousness.

The consciousness of the normal individual can
accept a number of impressions from the outer world,

the number varying according to the constitution of the brain and its training; but when his attention is strongly concentrated, he may become unconscious of everything except the matter having his attention. Such contraction of the field of consciousness is entirely normal. Abnormal, long-lasting narrowing of the field of consciousness is the condition of the hysterical, in which different sensorial impulses correctly sent in by the peripheral organs, and properly carried to the cerebral cortex by the nerve fibres, are not registered in the final layer of cells (psychologically considered) which subserve the purpose or are the organs of conscious perception. Just how the cells are out of function is unknown, but the neurone theory and recent observations on the activities of nerve cells permit the erection of a working hypothesis. We have only to assume that the dendrites of the cells in question have retracted, to understand that no impulse can be transmitted to them from the brushy endings of fibres carrying sensory impulses. The eye may be entirely normal, the optic nerve and optic radiations functionate perfectly, but if the cells serving the purpose of conscious vision are in the condition just assumed to exist, the individual cannot see, and is psychically blind. Similarly we may explain all the forms of anæsthesia that are found in hysteria.

In a somewhat analogous way, we may explain the paralyses of hysteria. If the large cortical cells of the motor areas are themselves considered as

under the domination of a higher layer (psychologically considered), subserving the purpose of conscious volition, we have only to assume the retraction of the dendrites of such higher cells to understand how a hysterical palsy occurs.

The normal mind, when in a state of concentrated attention to some one matter, is not conscious of ordinary sensorial impressions, nor at times of extraordinary ones. But such sensorial impressions are received by cortical cells, yet not transmitted to the higher (psychologically considered) cortical cells in direct relation to consciousness. Thus impressions are made upon the sensory areas of the cortex, yet below the cortical plané of consciousness; later, when the attention is no longer concentrated, such impressions may be and often are, passed on to the plane of consciousness and become recognized as past experiences.

Conversely, when consciousness is suspended as in the state of induced hypnotism, the lower cortical cells may be impressed by a command from without the subject, and both motor and sensory areas will respond accordingly; the former then produce spasm or paralysis in a limb or other part of the body, the latter cause hallucinations, either positive or negative in some of the senses. The state of being thus affecting different cortical areas, and through them the different organs of the body, is known as suggestibility. In the abnormal brain such suggestion can arise from within, but in the plane of sub-con-

sciousness, and is termed auto-suggestion. A state of double personality thus is seen to exist both in the ordinary hypnotic state and in the hysterical. To attempt any explanation of this fact would lead us into the regions of psychological speculation.

The logical faculty, that is the reasoning from premise to conclusion, is not the highest of our intellectual faculties. For, given a premise with a middle term, the conclusion can be made by a machine equally well with the brain cells. Indeed, the late Professor W. Stanley Jevons invented a " logical machine." *

Concerning it he says: " But when any proposition is worked upon the keys, the machine analyses or digests the meaning of it and becomes charged with the knowledge embodied in that proposition. Accordingly, it is able to return as an answer any description of a term or class so far as furnished by that proposition in accordance with the Laws of Thought." Some brain cells, perhaps some definite layers, thus digest information and attendant propositions brought into them, and finally present to consciousness results that are practically mechanical solutions of problems, in so far as consciousness has not taken cognizance of the cellular activity then going on. In the normal brain such results must be presented to consciousness and the critical faculty being then called into play, acceptance or rejection of the conclusion follows. In the abnormal brain

* *Vide* Principles of Science. Vol. I. p. 127.

consciousness is not coupled with power of decision;
and the conclusions are impressed, so to say, upon,
it may be, either the motor or the sensory cells of
the cortex. Jevons's machine acted infallibly: that is
to say, with the material proposition presented to it;
so do the cortical cells. But if the information sent
to the cortex be insufficient or false, then machine-
like, the cells will give a truthful conclusion, yet only
truthful according to the character and extent of the
information received. Hence, hallucinations, negative
or positive, being accepted as facts, mislead the
higher intellectual powers, especially that of judg-
ment or choice, into believing as true that which is
essentially false. The case of the mother, given by
Gowers, who, having struck her child, felt her arm
and hand immediately paralyzed, can be explained.
The swift conviction that she had done wrong and
that she might be punished by a paralysis, was a
sub-conscious result of the reasoning process, and im-
pressed itself at once on the related motor cortical
area. The process of reasoning or logical faculty
does not require for its correctness the presence of
consciousness; indeed, its anatomical instruments may
be likened to the parts of the Logical Machine.
When in the proper condition of readiness for work,
the machine " represents a mind endowed with
powers of thought, but wholly devoid of knowledge."
. . . . " But when any proposition is worked upon the
keys, the machine analyses or digests ; " " and is able
to return as an answer any description of a term or

class so far as furnished by that proposition, in accordance with the laws of thought." The machine requires an operator to see that false knowledge be not presented to it, and the normal processes of the brain in this department have to depend upon consciousness as the first guard against admitting false information. With the field of consciousness greatly restricted and suggestibility greatly increased, the thought that the arm that struck the child might be paralyzed in punishment is reasoned out, sub-consciously, as being true, and the cells of the related motor area are impressed accordingly, and paralysis occurs. The condition then is more than a delusion, since it involves something more than a false belief, for the paralysis is there and is just as real to the patient as is the blindness in hysterical amblyopia. To place the motor phenomena of hysteria in the same class as the sensory ones, the former might be called motor hallucinations, of negative type when paralytic, of positive type when spasmodic or contractured—the former term has however the disadvantage of being a paradox.

Paralysis of the Will has been held as the explanation of many of the symptoms of hysteria. Whatever we may consider the Will to be we must admit that it is operative only within the field of consciousness; hence when the latter is restricted or narrowed, the former is only active within such limits and then is only too assertive.

Organs which in the normal state act without our

conscious perception and beyond the domain of volition,
are frequently disordered in hysteria, such disorders
being part of the hysterical manifestation, but whether
through direct influence from the brain or not it is
difficult or impossible to decide. The digestive tract,
the secretory glands, the heart, the lungs, the vaso-
motor system and even the trophic functions, present
symptoms that at times may be alarming.

The mental powers of the hysterical are not changed
by the affection, but remain great or small according
to the original type of mental endowment; but hysteria
is found more often among those who are intelligent
(and frequently having the artistic temperament) than
in those who are on a low mental plane. The char-
acter, however, shows peculiarities. It is full of con-
tradictions. The inability to fix attention on anything
outside of themselves and their hallucinatory ailments,
makes them forgetful, inattentive, distracted. Trying
to convince them of the falseness of their sensations,
etc., makes them feel as martyrs, that they are mis-
understood, etc., and arouses a longing for sympathy
which in turn leads to exaggeration on their part and
often to intentional deception.

Hysteria is characterized by certain permanent
symptoms termed stigmata and, at varying intervals,
by episodical manifestations or paroxysms. The stig-
mata are anæsthesias, hyperæsthesias, paralyses and
contractures.

The anæsthesia is, in the majority of cases, a hemi-
anæsthesia and usually affects the left side of the body,

being limited by the mid-line. In other cases it may involve part of a limb or of the face. In any case its extent does not coincide with the distribution of sensory nerves and is mostly a loss of pain-sense, the tactile and temperature senses being only lessened, if at all affected. It commonly involves the mucous membranes within the affected area. It is a negative hallucination and its presence may be unsuspected by the patient until it is discovered by tests. It has been removed or even transferred in part to the other side of the body by the application of a magnet or of metals or of pieces of wood made to represent these (Charcot). Such removal lasts but a short time.

Anæsthesia of the retina is a frequent manifestation in hysteria in connection with hemianæsthesia. It is most often a concentric narrowing of the visual field on the affected side with a lessened narrowing on the other. The fields for colors are lessened in unequal degrees, those for yellow and blue being least affected. In complete bilateral amaurosis, which is the extreme manifestation of retinal anæsthesia, tests can hardly be expected to succeed, but in monocular affections of this kind, the stereoscope has shown that the patient really saw but was unconscious of the fact. Loss of taste, smell and hearing on one side is sometimes found.

Hyperæsthesias, usually in small insular areas may exist within the area of anæsthesia, but generally they are in certain regions, chiefly under the mamma, in the region above Poupart's ligament (the so-called ovarian region), over the upper end of the sternum,

and also over its middle. These spots are the seats of hypersensibility and often of painful sensations; pressure upon them, especially the ovarian one, may cause a spasm.

The paralyses of hysteria are hemiplegia, monoplegia, and paraplegia. No paralysis of an isolated muscle occurs and the face is but rarely, if ever, affected; but contracture of the muscles of one side of the face may by contrast give the appearance of paralysis of the other. The paralyses usually occur rather suddenly, after some mental or physical shock or after a spasm. In the hemiplegia the gait is unlike that of hemiplegia from organic disease; the patient does not "mow the ground", the knee bends somewhat and the foot rarely scrapes the ground.

Hysterical paraplegia is not an infrequent manifestation; it is often associated with the hyperæsthesia of "irritable spine" and thus may lead to the erroneous conclusion that disease of the spinal cord is present. It is in many instances attended by contracture.

Inability to walk or to stand (astasia-abasia) would appear to be an incomplete form, since the patient can move the limbs at will when in the supine position.

In relation to the paralyses, is the loss of power in arms or legs. Here the individual when walking loses the supporting power of the legs and may fall, or the arms act in a similar way and objects held in the hand are dropped. This condition is termed amyosthenia.

The most common paralysis of hysteria is that

known as hysterical aphonia due to paralysis of the
adductors of the vocal cords. The patient has no
voice for speaking yet can cough or occasionally can
sing, or during a dream can speak with ordinary
voice.

When the lips and tongue are affected—which is
not frequent—the patient cannot even whisper and to
this condition the term mutism is applied.

Hysterical contracture may affect the limbs of one
side, or one limb only or any part of it. It frequently
appears after some injury or after a convulsive seizure
or with hysterical hemiplegia. It develops rapidly,
unlike the contracture in genuine organic hemiplegia,
is more intense than the latter, while effort on the
part of the examiner to overcome it causes increase
of it that is readily felt.

The arm is most often affected; the upper half is
adducted, the lower flexed at the elbow, the hand
flexed at the wrist, the fingers flexed or clenched or
at times fingers and thumb are in the pen-holding
position.

In the lower limb the contracture is one of extreme
extension, the dorsum of the foot being in a line with
the tibia, while the toes may be contracted or hyper-
extended. In some cases the muscles become con-
tractured under slight provocation, such as pressure,
a slight blow, or even stroking the part. Such con-
dition is known as the contractural diathesis.

Tremor is frequently seen as a part of the hysteri-
cal condition. It may be of any variety, and at times

may show the intentional character. Ataxia, cerebellar in type, has been observed: so has athetosis.

Other symptoms, that need only to be mentioned, are: Globus hystericus, or a feeling as if a ball were rising in the throat, or at times as if the throat were constricted; vomiting, very frequent and often without accompanying nausea—the vomited matters may contain blood, and thus give rise to erroneous diagnosis. The readiness with which fæces and urine can be put into the vomited mass by a hysterical patient, makes it necessary to watch such patients narrowly.

Eructations, borborygmus, excessive flatus, meteorism stimulating tumor or pregnancy, are very common, while the latter may need examination under anæsthesia for detection and cure. Constipation is common and obstinate; anuria at times or polyuria may exist, and, as a rule, polyuria of almost colorless urine follows a hysterical attack.

Hysterical joint is most frequent in the hip or knee. The skin over and about the joint is extremely tender, the patient not tolerating the slightest touch, but the extent of pain is too great and the rigidity, when present, too extensive for a real arthritis. Some swelling may be present, however. Von Esmarch has stated that immersing the joint in warm water relieves the pain of a real arthritis, but aggravates that of the hysterical affection.

Vaso-motor disturbances in hysteria are many, but most of them are seen in other conditions also. Blue

œdema, a cyanotic and œdematous swelling of the extremities, is remarkable for not pitting on pressure.

Hysterical fever has been claimed to exist, but the possibility of deception here should be held in mind. On the other hand a lowering of the surface temperature in anæsthetic parts (especially with the blue œdema mentioned above) has been observed.

Attacks of coughing, lasting for hours, and obstinate hiccough, continuing in some instances for days, are at times seen in the hysterical.

Hysteria is separable into two forms, a minor one in which the severe convulsions do not occur and the permanent stigmata are not present. The emotional weakness, the hyperæsthesia of the sensory organs, the presence of globus or some similar sensation in the throat, the loss of self-control, the pains from which she can be diverted readily, the tender spots along the spine, the mental suffering at being "misunderstood," the exaggeration as to the severity and uniqueness of her symptoms—are some of the characteristics of the minor hysterical state. At times a hysterical chill with trembling, fits of uncontrollable and causeless laughter, alternating with paroxysms of weeping, equally causeless, appear when her wishes and projects are opposed.

In hysteria major the presence of the stigmata or some of them is found between the paroxysms, which may affect the larynx, stomach or diaphragm, causing symptoms already mentioned or which may consist of convulsive attacks.

The hysterical convulsion is brought on by some emotion, such as fright or anger, or from injury. The patient falls, yet usually without injuring herself; the breathing may be dyspnœic, but there is no arrest of respiration and so no asphyxia. There may be some tonic contraction of the limbs or even opisthotonos, but commonly there are purposive movements of arms or legs, or of the trunk and head. Striking and thrashing about, beating the head against the floor, thrashing the trunk up and down on the bed, and every possible kind of voluntary movement, may be carried out. Talking or screeching may be constant or occasional, or the vocal sounds of animals imitated. The eyeballs are at times converged, or turned upward or in some other direction. The pupils are dilated and are responsive to light, but the difficulty of examining them is very great. The tongue is not bitten, but the lip may be, and there may be foaming at the mouth. The patient may at times be seen to observe those about her.

As regards the length of the convulsive attack, it may be for a few minutes, or it may extend over hours. It may be cut short by holding closed the mouth and nostrils for twenty or thirty seconds, by dashing cold water on the face, or by pressure on the so-called ovarian region in women.

Of the severe convulsive seizures, called *grande hystérie*, or also (improperly) hystero-epilepsy, it may be said that they are not seen in this country.

There is a preliminary stage of headache, psychical
change, usually increased irritability, anxiety, etc.,
which lasts in some cases for several hours. Next
appears the aura, generally the globus subjectively
felt as starting from the ovarian region of the anæs-
thetic side. The patient falls, and apparently loses
consciousness, a stage of tonic spasm begins, followed
by clonic spasm—this stage lasting two or three min-
utes. Next comes the second stage, or stage of con-
tortion or *grandes mouvements.* The patient's body
becomes arched, resting on the soles and the top of
the retracted head, with changes of position, by which
the body below the shoulders is raised high up and
then brought down, or the body is thrown from side to
side or the limbs in active, extended movements in
different directions, the patient striking out at times as
if enraged, and howling, screaming, cursing, or weep-
ing and laughing. This stage lasts a few minutes
and passes gradually over into the next, that of emo-
tional or theatrical manifestations. Here the patient
assumes attitudes and facial expressions, exhibiting
the various passions, such as fear, rage, love, hate,
ecstasy, etc., apparently under the influence of suc-
ceeding hallucinations, the stage lasting about a
quarter of an hour. The fourth stage is but the
gradually disappearing echo of the previous one, end-
ing at times with a fit of laughing or of weeping; it
is termed the stage of delirium. The whole attack
may last half an hour or less. It can be evoked, at
least it could in Charcot's wards, by pressure on

the ovarian region, and when begun could be stopped by the same procedure.

The attack may be repeated many times. There is no increase of temperature, and after cessation of the attacks, there is often left a paralysis or a contracture.

A part only of *grande hysétrie* may appear. Following an aura, the hallucinatory stage may come on, or even a cataleptic condition or somnambulism, the latter in some instances continuing for days or weeks at a time. On returning to the more normal state, the memory of events and circumstances occurring during the somnambulic condition is gone, but in the latter the memory of previous attacks is retained. Thus there is a doubling of personality.

Sleep, apparently normal, may follow a convulsive attack, and may continue for many days.

The diagnosis of the ordinary hysterical convulsion from an epileptic seizure is not always easy. But usually the purposive movements, the absence of injury to the patient in falling, the unbitten tongue, the reaction to painful impressions (pricking the sole of the foot with a needle, etc.), and the relatively long persistence of the seizure, mark the case as hysteria. But it does happen that an attack of *petit mal* passes over into a hysterical one.

Women are more subject to the "disease" than men, in a proportion variously stated as ten to one or six to one. Its first manifestation is generally in the years about the time of puberty, fifty per cent.

of cases beginning between the ages of ten and twenty. Cases have been reported as beginning as early as the second or third year, but in children paralyses and contractures are less often attended by anæsthesia than in the second decade of life or later. Many symptoms of neurasthenia are present in the hysterical. The affection rarely begins after the climacteric.

Prognosis. The younger the patient, the better the prognosis of cure; children will "grow out of it," if the home life and school life are directed in a sensible way, under conditions of good hygiene, outdoor exercise, etc., the avoidance of much petting or well-meant but injurious sympathy. In the third decade of life, cases of unusual type, or varying much from the ordinary hysteria minor, are less hopeful of cure. The severe forms, those following trauma, and such forms as occur in the adult male, are difficult of cure; the latter do not show the changeability of symptoms usually seen.

The influence of suggestion in hysteria must be used as part of the "cure," and in fact many "operations" for the relief of the trouble may be justly said to act by suggestion. Many ill-smelling drugs, such as asafœtida, valerian, etc., which have a high reputation among old-school physicians, may be considered as acting in the same way.

On the other hand, the danger of suggesting new symptoms to the patient by a minute examination for anæsthesia or hyperæsthesia, or by searching for

some internal disease, especially of the generative organs, should be kept in mind, and such examinations made only when evidently necessary.

Hysteria, or a condition not to be distinguished from it in its minor phase, can be produced by the effect of physical or even emotional shock, as in the case of a railroad accident. The attempt to erect a new class of disorders, known as traumatic neuroses, has not been successful, and we can speak of traumatic hysteria or traumatic neurasthenia, according as the former state is present or absent.

The remedies useful in the treatment of the manifestations of hysteria are many. As a matter of experience, certain ones are very frequently called for. Of all, the most important is *Ignatia*, and next to that in the spasmodic manifestations are *Silicea*, *Moschus*, *Tarentula Hispania*, *Belladonna*, *Lachesis* and *Stramonium*. In general, besides the foregoing, *Lilium tigrinum*, *Sepia*, *Platinum*, *Pulsatilla*, *Valeriana*, *Nux moschata*, *Chamomilla*, *Lobelia inflata*, *Asafœtida* and *Sulphur* have repeatedly proved serviceable.

The hygienic measures mentioned under Neurasthenia will be applicable in hysteria.

Neurasthenia, Nervous Irritable Weakness,

as the French call it, is a wide-spread affection, especially a result of modern civilization or perhaps rather of its accompanying vices and excesses. It has seemed to increase enormously during the past quarter

of a century and as it is often due to an inherited
"weakness" of the nervous system, it may well be
that the preceding two generations have by their habits
(especially the use of tobacco and alcohol) poisoned
their own germ-plasm so that development in the
children is faulty and unbalanced, especially in the
higher physiological structures.

Other injurious causes in the ancestors than those
just named are insufficient food (as in states of poverty),
exhausting diseases (phthisis, cancer, etc.) and similar
conditions that must have a profoundly injurious
influence upon the being procreated during such states.

The disease can be acquired by a perfectly sound
organism as a result of insufficient food during child-
hood or by severity or cruelty at the hands of parents
or guardians during the same period, by the habitual
use of alcohol, the excessive use of tobacco, by sexual
excesses in the young, especially masturbation. Pre-
mature sexual development and desire must be looked
upon as a symptom of the neurotic state, but early
indulgence only intensifies and ripens the already
existing morbid condition.

The hysterical individual is almost always neuras-
thenic but the reverse is not the case, there being
thousands of neurasthenics who have no sign of hysteria.
But neurasthenia has its own psychosis—hypo-
chondriasis, which is perhaps even more difficult to
cure than hysteria.

Neurasthenia may begin at almost any period of life.
It may be seen in young children or even appear first

in old age, but it generally is observed in the third decade, and the proportion shades away in both upward and downward directions.

The symptoms, although generally alike, can be classed under three heads: cerebral, spinal and sexual. In all cases, the symptoms can be described first as hyper-excitability, next as exhaustion. The first makes him hurry; he is under a sense of obligation to hurry; he hurries at his work and has to stop because of not being able to do the work at the same *tempo* as he feels urged, and so he becomes wearied or tired and "gives up," thereby causing annoying self-objurgations. He eats in a hurry, he is overcome by the sense of being hurried; it might almost be said that he sleeps in a hurry.

He is fearful. Apprehensions that he will fail accompany him in everything that he undertakes, or he refuses to undertake anything lest he should fail. His mind is distracted; he cannot study; his concentration is upon himself and his weakness; one must be free from extraneous thoughts in order to study but his mind has the thought that he is studying—and that fills his mind and so he does not take in what he is reading. He directs a letter and after posting it, does not know that it was properly directed—and he worries accordingly; he locks his front door and yet not knowing surely that he has done so, goes down again to make sure—and repeats this time after time. His sleep becomes broken and is not refreshing, or he is unable to sleep. He has headaches, most frequently

a sense of pressure on the vertex and often described
as a tight metal cap; similarly the sensation may be
a tightness (or at times the sense of bursting) in the
occipital region. Such symptoms give rise to the fear
of some organic brain trouble. Slight dizziness with
obscuration before the eyes or passing attacks of vertigo
increase his fears. In many cases other morbid fears
are present and make life a burden to him. The fear
of going into or crossing open spaces (agoraphobia);
of being in a closed room (claustrophobia); of being
alone, of looking down from high places, etc. are but
a few. To attempt any of these things, brings on an
attack of nameless terror that simply prevents the
continuance of the attempt and leads to refusal to leave
home or to go to church, etc.

Of the organs of special sense, the eye and ear
are extremely irritable. Photophobia is often present;
the eye muscles tire easily, the letters run together
after reading a short time, or vague visceral sensa-
tions are experienced (chiefly abdominal), sparks and
stars flit before the eyes, and *muscæ volitantes* are com-
mon annoyances. The patient is often acutely sen-
sitive to noise, probably from cortical irritability, as
it is noted even when the drum-head is thickened
and when evident lessening of hearing power is
present.

The general muscular power may or may not be
lessened, but is often exhausted after relatively
slight effort, especially in walking. Tremor is fre-
quently observed.

The abdominal organs may be affected. Nervous dyspepsia is a common complaint among neurasthenics; the liver acts imperfectly; intestinal fermentation may be excessive, intestinal activity lessened and constipation exist. The urine is, in many cases, concentrated, sp. gr. 1030 or over, is very high colored, small in quantity, and at times irritating when passed.

The sensory disturbances may be tingling or prickling of hands or feet, pain at the nape of the neck, described as aching or at times a sense of weight, pains along the back, bruised feeling of the scalp, aching of the limbs, often worse in the morning when lying awake in bed.

Cardiac symptoms may be present. Nervous palpitation, with or without acceleration of the pulse-rate, attacks of præcordial anxiety and pain, simulating angina pectoris, are not infrequent manifestations.

Vaso-motor weakness or irritability is seen in many cases. The feeling of increase of blood within the head, with redness or flushing of the face (often seen in women at the climacteric), with cold extremities and sweaty palms and soles, the easy production of red spots on the skin after pressure and lasting many minutes, are instances of the disturbance.

The sexual organs are to the patient, in many cases, the centre around which all his nervous symptoms revolve. Previous masturbation or other sexual excesses give rise to weakness, shown in frequent nocturnal seminal emissions. These cause worry and appre-

hension of incurable disease, or of permanent impo-
tency, or keep before the mind his self-reproaches·
In the married, inability for the sexual act causes
similar hypochondriacal fears.

The frequent recurrence of nocturnal emissions in-
tensifies the nervous state and causes great mental
depression. When the genitals are specially weak,
discharge of prostatic .or seminal fluid may occur
during stool, at times containing spermatozoids. *Im-
potentia coeundi* is not an uncommon complaint, but
is probably due, in a large proportion of such cases,
to the fear of failure.

Sleeplessness is quite a common symptom. It is
rarely absolute, and indeed the statements of the
patient in this regard must be received with a good
deal of allowance. The sleep is undoubtedly light,
and annoying dreams add to the trials of the patient.
A special feature related to vaso-motor aberration is
that the patient is very drowsy while up and about,
but on lying down becomes wide awake.

Pains in the back, often a sense of tiredness or
aching, unconnected with organic disease of any kind,
are not infrequent. The most common manifesta-
tion, however, is tenderness of the vertebral spines
to pressure, usually in the cervical region; with this
objective tenderness (or without it) there is a sense
of pressure in the post-cervical region, often extend-
ing upward into the occiput. The symptom may be
so severe and so constant as to rob the patient of
the power of attention to anything else.

In the neurasthenic the tendon reflexes are exaggerated. In most cases the patients are fairly well nourished.

The nature of the disease has been considered already, but the internal or external causes that keep it actively manifest are matters of speculation. The lithæmic theory is perhaps better than any other, but there are probably present in the system other peccant matters than uric acid.

The prognosis of neurasthenia depends first upon whether the condition is inborn or acquired, next the age of the individual and the length of time the symptoms have existed, and lastly the special form of the dominant symptoms. The morbid fears may be so intense, and the mental depression so great, that the case really is one of mental perversion or insanity, and should be treated as such. When the sexual symptoms are the most prominent, a real cure cannot be expected if the trouble has been the outcome of years of sexual excesses.

In most cases of neurasthenia so much improvement can be effected that we may speak of a relative cure; but it must be held in mind that, even then, influences injurious to the nervous system can renew the old symptoms.

Diagnosis is chiefly to exclude organic disease of heart or other internal organ, or of the central nervous system. The absence of organic signs would be conclusive in one direction, but their presence would

not prove the non-existence of neurasthenia, since the latter can also be present.

The hypochondriacal tendencies may lead to a real hypochondriasis, but this is to be distinguished from hypochondriacal paranoia.

Hysteria and neurasthenia often co-exist, but in neurasthenia there are not the stigmata of the former disease.

The treatment of neurasthenia is divisible into two kinds, hygienic and medicinal. Whether the lithæmic theory be correct or not, certain it is to the writer's mind that increased oxidation and tissue metabolism lie at the foundation of the successful treatment of neurasthenia. To effect this, hard physical labor (except in those whose work is such), gradually increased from a few minutes at first up to one hour a day; wood-chopping and sawing, rowing (when place and season permit) are the most efficacious kinds of labor. Next is bicycling, with its wonderful influence in developing the heart and lungs, provided the rider gradually inures himself to the new demands made upon these organs. The exercise should be carried out daily, with perhaps an omission of one day in seven; at its end a rubbing dry with a soft towel, and complete change of clothing and resting on a couch for an hour should follow.

Lessening the amount of nitrogenous food, with increase of vegetables and fruits, should be enjoined (but most neurasthenics seem dependent on a plentiful meat diet); as water is a necessity to facili-

tate tissue changes and elimination of excrementitious products, the use of drinking water between meals, is an important part of the "cure." Tobacco, tea and coffee should be eschewed, or, if this is felt to be impossible, should be used only in very small amounts. Alcohol in any form should be avoided.

The neurasthenic whose daily work is fatiguing may find the hard work prescribed above to be too exhausting; here the bicycle is of especial value. The writer has known more than one case in which fatigue from walking and standing (in an office or shop) has been entirely removed by an hour's ride on the wheel through the park or country.

Cold water to the spine, by means of a hose attachment with sprinkle-nozzle, has often an invigorating effect. It may be used at such time as the patient finds it to act best. In the morning it will dissipate the achy, tired feeling that so frequently follows the sleep of the neurasthenic, or if used just before retiring it may ensure better sleep.

Change of climate may be advantageous if business and other cares can be left behind; travelling, with its hurry and annoyances, must have a bad effect.

The drugs used in the treatment of neurasthenia are, as might be supposed from the variety and distribution of the symptoms, simply legion. Where the cerebral symptoms are dominant, with inability for mental labor, *Picric acid, Calcarea carb., Kali phos., Nux vomica, Gelsemium, Phosphoric acid, Phosphorus* are called for.

When the hypochondriacal tendency is marked, *Aurum, Kali brom.* (in a potency`, *Sulphur, Natrum mur.*

When insomnia is a chief symptom, *Ambra, Arsenicum, Cimicifuga, Coffea cruda.*

When the sexual organs are markedly affected, *Selenium, Picric acid* or its zinc salt, *Phosphoric acid, Nux vomica, Lycopodium, Agnus cast., Gelsemium.*

In general, *Physostigma, Berberis, China off., Plumbum, Silicea, Piper methyst.,* and other remedies have proven of value in the treatment of neurasthenia by the writer. It is advisable to take a full record of all the symptoms as given by the patient, and although a succession of remedies will probably be required to influence a case of the disorder, yet the proof shown by the record of the disappearance of certain symptoms or groups of symptoms will encourage greatly both physician and patient.

Traumatic Neuroses.

The effects of injury to the individual by trauma, especially in conjunction with shock, either mental or physical, has long been known to set up a train of symptoms not to be accounted for by the actual physical injury or indeed in the absence of any demonstrable injury. Such were often mistaken for cases of malingering.

Too many cases, however, appeared where no possible reason for simulation was present, yet in which the constancy or even worsening of the symptoms excluded such a view.

The effort was later made to consider the symptom-complex as one *sui generis* under the title traumatic neurosis, Charcot meanwhile labelling such cases "traumatic hysteria".

At present the most general view is that from trauma of any severe kind especially when sudden and accompanied by mental shock or fear (at times without such) there may develop symptoms of neurasthenia, hysteria, hypochondriasis, melancholia, etc., at times with almost complete loss of emotional control and of mental or physical capacity for the daily occupation.

In Germany the subject has received great attention because there a workman who is incapacitated for labor receives (partly from the State) a certain sum regulated according to his complete or incomplete incapacity, and hence every effort is made by physicians and jurists to expose attempts at simulation. In this country no such arrangements exist, and claims for damages against railway companies, manufacturing concerns, municipal corporations, etc. are made for injuries received, and are often defended by denying all liability unless objective signs of bodily injury can be proven to exist.

Most observers admit that in the hysterical and related states there is a tendency in the patient to exaggerate the symptoms, and that in traumatic hystero-neurasthenia such exaggeration is often found, but that this is not simulation.

The examiner of a case of nervous symptoms following any kind of accident should be extraordinarily

careful in making up his opinion and in fact should not do so until after a second or third examination at considerable intervals; and he should be extremely reserved in expressing his opinion in general or even in commenting on (in the presence of the patient) the results brought out by his tests. Suggestibility exists in the hysterical condition and is not wholly absent from the neurasthenic; so that the various and sometimes complicated methods of testing may well arouse the patient's attention to the highest degree and lead him to consider his case to be extremely grave.

The symptoms vary in different cases. In some the mental effort becomes soon exhausted or causes vertigo or vertex headache (metal cap sensation very often); the emotional control is lessened, the patient cries at the least cause or is worried beyond reason at his inability to earn wages. Mental depression is frequently present. The physical powers may show too great exhaustibility so that a little effort fatigues him.

The spinal symptoms may be predominant and the picture then is that of the long-known "concussion of the spine." Pains in the back, in the sacral region, in the cervico-occipital region, are the chief manifestation. The pains are great upon movement, and the patient keeps as nearly motionless as possible. Paralytic conditions, hysterical in character, may result, and may be paraplegia, hemiplegia, or monoplegia. If hemiplegia exist after injury to the head, it is always

on the side of the injury, thus establishing its psychical origin.

Anæsthesia, more often hypæsthesia, upon the side or part injured, or pains and paræsthesias of different kinds are frequently observed.

Concentric contraction of the visual field, bilateral, but most on the side predominantly anæsthetic, is the sign of a functional neurosis, whether traumatic or idiopathic in origin; the contraction should be of at least 10 degrees to be worth considering, and the field for colors is more contracted than for white. Simulation is, however, not impossible here and when suspected the field should be taken at different distances.

Certain symptoms have been found so frequently in conditions following accident that Rumpf considers them of diagnostic value. The first is a peculiar fibrillary tremor that spreads over the paretic muscles or may be more widely spread. It is seen after exposing the part to cold, after muscular effort and especially after applying a strong faradic current. The appearance produced is likened by Rumpf to the waving of a field of corn under the action of the wind. It is not always to be elicited and may exist in progressive muscular atrophy.

Mannkopf's symptom is an acceleration of the heart's action when firm pressure is made on some painful spot. It is not always seen, and Rumpf has at times found that the pressure causes retardation and irregularity of cardiac action. In testing in this way the patient must breathe with his ordinary regularity; if

excited and the respiration becomes rapid, a corresponding change will occur in the heart's action.

Vaso-motor changes are often present; among them are cyanosis, which may be extensive in distribution or in limited areas, a ready blushing of face, neck, even the nape or over the chest, and urticaria factitia.

The vaso-motor symptoms, Mannkopf's and Rumpf's symptoms, the contraction of the visual fields (when repeatedly and properly tested), are beyond simulation and hence are of great importance.

Jessen has described a form of tremor following trauma,* in which the lower extremities are affected by a convulsive tremor when motion is attempted. The muscles are strongly contracted, hard and prominent, and on cessation of effort they relax with difficulty. Jessen terms the condition "spastic tremor-neurosis." In some cases no other symptom is present, but in others psychical anomalies were observed; in one case (Nonne) attacks of polyuria and tachycardia occurred.

The symptoms of the traumatic neuroses may appear immediately after an accident or injury, or may not become manifest until some possibly trifling bruise or slight dislocation, etc., has been remedied. In other cases, weeks or even months may pass before the symptoms become evident. In some cases the disturbances of sensation, contraction of the visual field, increase of the reflexes, existed unknown to the subjects, and their origin could only be ascribed (by

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* Pseudo-spastic paresis with tremor: Fürstner.

means of the history) to more or less severe accidents
through which the individuals had passed and of which
they no longer thought. The condition may continue
for years or permanently, without change, or symptoms
may develop in course of time showing that in addi-
tion to the functional disturbance of the central ner-
vous system, organic changes have occurred there.

Prognosis. In light cases which are general in
extent, complete. cure may be looked for; when there
is much psychical change or when the neurosis is a
local one or when the cardiac innervation has suffered,
the outlook for cure is not bright. Oppenheim con-
siders that the paralyses and sensory disturbances are
far more obstinate than the corresponding symptoms
in hysteria.

In any case the existence of a suit for damages or
claims for accident insurance, militate greatly against
improvement, by the anxiety of the patient concerning
his family's future or his own. A favorable settlement
in such cases may be followed by marked improve-
ment, and this was formerly taken as evidence of
simulation or at least of intentional exaggeration; but
in view of the many cases in which full damages
or insurance were allowed, no improvement followed,
the argument falls to the ground.

The treatment of the traumatic neuroses must be
psychical, hygienic and medicinal. In the former,
encouraging the patient in every way to resume his
occupation as soon as the physical results of his injury
have passed away. In the second, the measures already

prescribed under Neurasthenia will be found here equally applicable. And similarly for the third, except to add that *Arnica* given internally has at my hands been of great service—in the effects of shock from the electric current it is, I think, the main remedy.

Occupation Neuroses.

The repetition of muscular effort, especially when the smaller muscles are for long periods engaged in carrying out finely co-ordinated movements, or larger ones are compelled to make with great rapidity the same movement, appears to give rise to a condition of irritability in the nervous system that finally abolishes the ability to perform the action in question, by setting up spasm or pain or both. The sufferers from the affection are usually neurotic, the neuropathic state resulting to a great extent from their confined mode of life, or from injurious influences, such as excessive use of alcohol, tobacco, prolonged anxiety, debilitating effects of illness, etc.

The most common manifestation is the **writer's cramp.** It appears in those who do more or less continuous writing, but it is believed that the inimical influence is rather in the mode of handling the pen than in the amount of the daily task. The movements of the pen, when caused by the action of the fingers alone, or of the fingers and hand, induce far greater fatigue in the small muscles of the hand than do the movements of the arm and forearm muscles in them, when writing is done in the so-called free-hand

mode. Writing under conditions of insufficient space has in one instance caused the affection, the case being that of a medical student taking notes upon his knee, where the finger movement only could be employed.

The trouble begins gradually, the victim finds that he does not write so fluently as usual; later, he is conscious that he is grasping the pen-holder tightly and this, it seems to him, causes unusual fatigue of the finger and thumb muscles or in those of the whole hand. Later still—it may be months—distinct spasm affects the thumb and the next two fingers and the movements of the pen cannot be controlled; in some instances the spasm is in the extensors of the fingers and then the pen-holder cannot be retained in the position for writing. Finally, the muscles of the fore-arm may become involved in the spasm.

In some cases the painful sense of fatigue increases in a greater ratio than does the spasm and becomes a distinct separate sensation of pain in hand or wrist, at times continuing for hours afterward, or it may extend up the arm.

A rare form of the affection is the occurrence of tremor of the hand and arm when writing; another is the so-called paralytic form, in which the muscles become incapable of action when the attempt is made to write.

The disease is difficult of cure if it has been of long standing.

Sufferers from the disorder can usually employ the hand at other occupations without trouble of any kind.

When the left hand is made to do the work of writing, after the right has become useless for this purpose, it, too, is apt to become similarly affected.

The seat of the disease is the central nervous system, but whether the motor cells in the cervical segments of the cord or those of the cortex are most affected cannot be readily decided. The pain sensation may be due to changes in the nerves of the arm and hand akin to those in neuralgia. Neuritis is said to have been observed in some cases, but such are not to be classed with genuine writer's cramp.

Treatment must begin with complete cessation of writing; if this be impossible then the mode of writing must be changed, so that the movements in writing are made from the shoulder, the hand taking but little, if any, part in the act; great assistance may be derived from using one of several mechanical devices for attaching the penholder to the writing fingers, thus permitting the pen to be moved across the paper without effort on their part. The use of gold pens, and of thick penholders, covered with rubber at the clasping point, are believed to be helpful in lessening the tendency to spasm. Massage, or a combination of massage and gymnastics, has many advocates, while galvanism given daily (5—10 ma., cathode labile along nerve trunks, beginning in upper arm, anode beside cervical spines) has been highly praised. Both Gowers and Oppenheim think it of doubtful value. The hygienic measures recommended in the treatment of neurasthenia should be employed here.

The other occupation neuroses make a long list. Among them piano-player's, violinist's, telegrapher's, are those chiefly seen. The underlying conditions in the production of these affections are the same as in writer's cramp, and the principles of treatment are the same as in that trouble.

Akinesia Algera. Under this term has been described a nervous affection, in which motion becomes impossible, owing to pain caused thereby. The condition is not a disease, but a symptom-complex, and is found in those of intensely neurotic constitution. The patient finally becomes completely bed-ridden, and is as if paralyzed. Even passive motion becomes painful, and moving the eyes or the jaws (as in eating) is provocative of pain. There is a tendency to insanity, usually of depressed type. The disease has been considered incurable, but Erb has recently reported the cure of a case that had been bed-ridden for nineteen years, the cure having been made by suggestive influence and by gradually encouraging the patient to use his limbs, at first but little, and then increasing the extent of activity, but always in spite of the pain. It took some two years in the process of cure. The medicines given were only to strengthen the general body functions.

Nervous Affections depending upon Diseases of the Thyroid Gland. The real function of the thyroid gland is as yet unknown, but when the gland is removed certain symptoms appear, characteristic of the diseases, tetany and myxœdema. The condition fol-

lowing complete removal of the gland is one of mental dulness, sluggishness of physical actions, slowness of body growth, thickening of the skin, and, as final terms, marasmus and imbecility. A form of sporadic cretinism has been observed, due to myxœdema, and the endemic form may be the result of degeneration of the gland in spite of its enlargement.

When the thyroid gland is too active, a set of symptoms follow, which are in many respects the opposite of those observed in myxœdema, the excess of secretion acting as a poison. Such symptoms can be produced in the healthy by administering thyroid extract, but, as the extract when given to a case of myxœdema relieves the symptoms, the ordinary idea of poison is not to be held.

Exophthalmic Goitre. Basedow's Disease. Graves' Disease. The disease is seen most often in young persons, few cases occurring after the age of forty; it is far more frequent in women than in men. The patient is often of nervous constitution, irritable, with nervous disease among relatives. Several cases have been in the same family.

The exciting cause most frequently assigned is violent mental emotion; next in frequency are physical over-exertion and a preceding illness.

The first symptoms are those of general malaise, feeling of weakness, irritability, often sleeplessness. The chief symptoms are acceleration of the heart's action, with increased pulsation in the arteries; tremor,

resembling that following excitement; increase in size of the thyroid gland; and protrusion of the eyes or exophthalmos.

The symptoms just given are not always present, but the first and second usually are.

The heart-beats may be as high as 180 per minute, and often 140 or 150; under mental or physical excitement the rate may increase. The patient may suffer from palpitation of the heart, and be conscious of pulsation in the carotids, abdominal aorta, etc., while the pulsations of the carotids are often distinctly visible.

The tremor is a rather rapid vibration, 8 or 10 oscillations in the second, and with only slight variation from muscular or mental effort. A sensation of general internal trembling is felt in many cases and is part of a general "nervousness," characterized by irritability, apprehensiveness, restlessness, etc.

The increase in the size of the thyroid gland is not due to hypertrophy of glandular tissue, but to vascular engorgement, or better described as a kind of erection; a bruit can be both felt and heard (on auscultation) in the enlarged gland. Both lateral lobes are usually affected and generally equally; the middle lobe is usually not much enlarged until the disease has lasted a considerable time. One lobe only may be affected, and then the exophthalmos may be unilateral.

Exophthalmos is, if present, bilateral as a rule. The eyeball does actually protrude, perhaps owing

to vascular dilatation of blood vessels within the or-
bit and to some increase of intra-orbital fat. The
eyelids are often more or less retracted, increasing
the width of the palpebral fissure; at times the up-
per lid is retracted so much that a strip of white
sclerotic is visible above the cornea (Stellwag's symp-
tom); in other cases the upper eyelid lags when
the patient looks down, and for the time being the
sclerotic is seen above the cornea (Graefe's symptom).
The retraction of the upper eyelid may be masked
when œdema is present, as it occasionally is. In
some cases complete closure of the lids is impossible.

Other symptoms considered as subordinate to those
already given, are nevertheless of great importance.
A very annoying one to the patient is the subjective
sensation of heat; it may come in flushes that seem
to overpower with a sense of suffocation; it may be
followed by an outbreak of sweat. The skin is com-
monly reddened and moist, often indeed bathed in
sweat; hence its normal resistance to the passage of
the galvanic current is notably lessened, frequently
by more than one half.

The skin in different parts of the body may be
pigmented with chloasma-like spots; the body often
wastes, and the wasting, together with the discolor-
ation of the skin, gives a cachectic appearance to the
face—the wasting may cease, and later be replaced by
marked increase of tissue. Insomnia is observed,
complete or partial, in many cases.

Often present, yet not characteristic of the dis-

ease are, sudden weakness of the legs, insufficiency of convergence of the eyes, causeless outbreaks of vomiting or of diarrhœa, polydipsia and polyuria. The knee jerks may be normal, exaggerated, or even wanting.

Hysteria, or some other nervous affection, may be present as a complication, and dilatation and hypertrophy of the heart have been noted in the course of the disease.

The prognosis is fairly good for cure in cases of not great intensity; remissions or imperfect cures are most often seen. When the disease has lasted for years, when the heart shows distinct evidences of being affected, when the mind is greatly affected, when exophthalmos is very marked, when the thyroid is very large, and when the pigmentation of the skin is intense and widespread, the prognosis for cure is not so favorable. Death comes from exhaustion, from heart affection, or from some intercurrent disease.

The diagnosis is to be made by the four cardinal symptoms, but not all are necessarily present in the same case. Exophthalmos is frequently absent, and less frequently the enlarged thyroid. Most observers hold that tachycardia with the tremor and some of the subordinate symptoms are requisite for the diagnosis. The writer has seen a case of undoubted Basedow's disease in which the pulse was only 72, and Oppenheim points out that the acceleration of pulse rate is not necessarily continuous, that it has periods or times of remission.

Real hypertrophy of the thyroid gland may give rise to many of the symptoms found in Basedow's disease, from pressure on the sympathetic. The disease may last for years.

The homœopathic treatment of exophthalmic goitre has wrought many cures. The writer has seen cures from *Lycopus*, and prefers it as a routine remedy in the disease. A case was greatly improved under *Sparteine sulphate*, 3d dilution, and, accidentally learning the name of the remedy, the patient no longer returned, stating to another patient that he could get the remedy and treat himself thereafter.

Cures have been reported by *Bromine*, and great improvement from *Aurum, Calc. carb., Iodium, Ferrum, Natrum mur., Cactus* and *Belladonna*.

Galvanism, the poles being placed just behind the posterior borders of the sterno-mastoid muscles and an inch above the clavicle, is always employed by the writer. The application should be once a day, the current strength from 3 to 5 m-a, the length of each sitting from 5 to 10 minutes. In most cases the pulse rate falls from 10 to 20 beats, and if the fall is nearing 20, the current should be lessened in strength. The current strength should be gradually increased from zero, and at the end of the sitting should be gradually decreased to zero before removing the electrodes.

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